University of Alberta

Characteristics of Patients Who Achieve Successful Cardiovascular Risk Reduction

by

Sandra Engi

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Nursing

Faculty of Nursing

Edmonton, Alberta
Fall 2006

Library and Archives Canada

Published Heritage Branch

395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque et Archives Canada

Direction du Patrimoine de l'édition

395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file Votre référence
ISBN: 978-0-494-22206-5
Our file Notre référence
ISBN: 978-0-494-22206-5

NOTICE:
The author has granted a non-exclusive license allowing Library and Archives Canada to reproduce, publish, archive, preserve, conserve, communicate to the public by telecommunication or on the Internet, loan, distribute and sell theses worldwide, for commercial or non-commercial purposes, in microform, paper, electronic and/or any other formats.

The author retains copyright ownership and moral rights in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

AVIS:
L'auteur a accordé une licence non exclusive permettant à la Bibliothèque et Archives Canada de reproduire, publier, archiver, sauvegarder, conserver, transmettre au public par télécommunication ou par l'Internet, prêter, distribuer et vendre des thèses partout dans le monde, à des fins commerciales ou autres, sur support microforme, papier, électronique et/ou autres formats.

L'auteur conserve la propriété du droit d'auteur et des droits moraux qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

In compliance with the Canadian Privacy Act some supporting forms may have been removed from this thesis.

While these forms may be included in the document page count, their removal does not represent any loss of content from the thesis.

Conformément à la loi canadienne sur la protection de la vie privée, quelques formulaires secondaires ont été enlevés de cette thèse.

Bien que ces formulaires aient inclus dans la pagination, il n'y aura aucun contenu manquant.

Canada

Abstract

Investigation and treatment of risk factors for cardiac disease have been identified as important components of cardiovascular care. The purpose of this study was to identify patient characteristics that independently predict successful cardiovascular risk reduction. Success was measured as the attainment of target LDL levels. A retrospective cohort design was used to examine data from the Cardiovascular Risk Reduction Clinic at the University of Alberta Hospital. Logistic regression modeling was used to test independent association of predictor variables (gender, smoking history, Framingham risk category, family history, number of visits, prevention, base LDL on admission already at target, age, BMI). The results of logistic regression modeling indicated that male gender, a low-risk Framingham, a BMI of ≥ 30 kg/m^2, the number of visits, and already being at target LDL on admission were significant independent predictors of the ability to attain target LDL levels.

Acknowledgements

I would like to thank a wonderful group of colleagues for their tireless and unyielding support of this project, for without their belief in the purpose, this thesis would never have been completed. Thank you to Dr. Glen Pearson for opening up his clinic and knowledge base to allow me to gather data and improve my personal knowledge on risk reduction management, to Dr. Michelle Graham for believing in my capabilities and supporting me through this project and to Dr. Beverly Williams for her persistence and support throughout this entire educational journey. Finally a special thank you to Dr. Colleen Norris, whose belief in my abilities and support have helped me to achieve this goal, for without her support, this thesis would never have been completed. To my editor, Linda Pasmore, who has diligently edited various works, thank you. Last but not least, I would like to acknowledge my family and friends who have supported me emotionally throughout this journey.

Table of Contents

CHAPTER 1 INTRODUCTION 1

Purpose 1

Research Question 2

Problem Statement 2

Significance of the Study 2

CHAPTER 2 LITERATURE REVIEW 4

Risk Factors and Risk Factor Reduction 4

Nonmodifiable Risk Factors 5

Modifiable Risk Factors 7

Framingham Risk Scoring 17

Primary and Secondary Prevention 18

Cardiovascular Risk Factor Reduction Clinics 19

Conceptual Framework for Behavior Change Related to Risk Reduction 20

Characteristics of Patients Who Achieve Successful Risk Reduction 24

Conclusion 27

CHAPTER 3 METHODS 29

Sample 29

Objectives 31

Primary Objective 31

Secondary Objectives 31

Outcome Variable 31

Predictor Variables 31

Coding of Variables 32
Analysis 36
CHAPTER 4 RESULTS 38
Descriptive Analysis 38
Sociodemographic Variables 38
Clinical Variables 41
Objective 1 43
Hypothesis 1 43
Objective 2 48
Hypothesis 2 48
Objective 3 48
Hypothesis 3 48
Primary and Secondary Prevention (Subgroup Analysis) 49
CHAPTER 5 DISCUSSION 52
Gender 53
Framingham Risk Category 54
Number of Follow-Up Visits 55
Obesity 56
Primary and Secondary Prevention 57
Clinical Implications 58
Obesity Reduction 58
Smoking Cessation 59
Limitations 60

Research Implications 61

CHAPTER 6 CONCLUSION 62

REFERENCES 65

APPENDIX A: TRACKING CARDIOVASCULAR RISK FACTORS 71

APPENDIX B: CARDIOVASUCLAR RISK REDUCTION CLINIC DATA SHEET .. 72

APPENDIX C: REQUEST FOR ETHICS COMMITTEE APPROVAL 73

APPENDIX D: CARDIOVASCULAR RISK REDUCTION CLINIC FOLLOW-UP FORM 78

APPENDIX E: DETERMINATION OF FRAMINGHAM RISK SCORE 79

APPENDIX F: FLOW CHART OF CASE EXCLUSION WITH NUMBERS AND RATIONALE 80

List of Tables

Table 1. Method for Calculating the 10-Year Risk of Coronary Artery Disease in a Patient Without Diabetes Mellitus or Clinically Evident Cardiovascular Disease Using Framingham Data 35

Table 2. Frequency Table for Independent and Dependent Variables 39

Table 3. ANOVA Table of Comparison of Means to Meet Target LDL 44

Table 4. Crosstab Comparison Variables With Met Target LDL 45

CHAPTER 1

INTRODUCTION

Cardiovascular disease (CVD) burdens our society with the ongoing high cost of management in both financial terms and quality of life. Several risk factors have been identified that can be used to predict and manage CVD. The characteristics of some of these risk factors are modifiable with lifestyle changes or pharmacologic therapy. Population demographics, including age, gender, family history, education level, and socioeconomic status, can be used to predict the prevalence of risk factors. Risk-factor control has come into focus in health care over the last 10 years with regard to reducing the rising incidence of CVD. The focus has now turned to specialty clinics that manage people identified with the highest risk of CVD, particularly those clinics that focus on reducing risk due to hyperlipidemia. Many of these clinics utilize the transtheoretical behavior change model[1] in delivering effective programming. However, there is a gap in the literature in the descriptions of patient characteristics such as age, gender, socioeconomic status, and education levels that may predict successful cardiovascular risk reduction.

Purpose

The purpose of this study is to explore the relationship between patient characteristics and successful risk reduction among patients who attended a lipid reduction clinic at the University of Alberta Hospital between 2000 and 2005. As well descriptive analyses will be performed to determine the association between changes in body-mass index (BMI) and low-density lipoprotein (LDL) reduction.

Research Question

What are the patient characteristics of successful cardiovascular risk reduction? This question was explored to address gaps in the literature and to describe patient characteristics that may correlate with successful cardiovascular risk reduction.

Problem Statement

In order to decrease the burden that CVD has on society it is imeritive that the health care system develop strategies to decrease the effects of CVD. Several risk factors have been identified that can be used to predict and manage CVD. The characteristics of some of these risk factors are modifiable with lifestyle changes or pharmacologic therapy. Risk-factor control needs to remain a primary focus of prevention in order to stem the rising incidence of CVD. The focus of risk reduction has turned to specialty clinics that manage people identified with the highest risk of CVD, particularly clinics that focus on reducing risk due to hyperlipidemia. However, there is a gap in the literature in the descriptions of patient characteristics that may predict successful cardiovascular risk reduction. Identification of characteristics that may predict successful risk reduction is helpful to program planning. The literature identified this area as requiring further research and investigation to develop and target programming toward those individuals who are not benefiting from current risk-reduction strategies.

Significance of the Study

According to the Heart and Stroke Foundation of Canada (HSFC),[2] the number of cases of CVDs over the next 20 years will steadily increase and thus result in an increased burden on society. Billions of dollars are spent each year on treating CVDs, and they are the major causes of hospitalization of men and women and one of the most

costly contributors to both direct and indirect health care costs in Canada. A wide variety of factors interact to influence health, such as income, gender, and education level. As income and education level increase, the prevalence of improved lifestyle choices also increases. The modifiable risk factors for CVD are largely preventable and are primarily a consequence of lifestyle choices; therefore, decreasing the prevalence of these risk factors can decrease the overall burden of heart disease.[2,3] Approximately 45% of the current reduction in mortality is attributable to an improvement in medical therapies for coronary disease; the remaining 55% results from risk-factor reduction. The aim of this study was to explore the relationship between patient characteristics and successful risk reduction among patients who attended a lipid reduction clinic at the University of Alberta Hospital between 2000 and 2005. The focus of the literature has been on recommendations for the prevention and treatment of risk factors, and this study examined the real-life application of such strategies.

CHAPTER 2

LITERATURE REVIEW

The literature review revealed several key features of cardiovascular risk reduction. Researchers discussed the enormous burden on society, identified the key risk factors, and explored their prevalence according to gender, age, education level, and income level. Several authors emphasized the importance of reducing these risk factors, defined set target goals, suggested reduction strategies, and addressed the weaknesses of current risk-reduction management strategies. These researchers also made recommendations and discussed the benefits of risk-reduction strategies available through specialty clinics that focus on behavior change as a key component of risk reduction and utilize the transtheoretical behavioral change model.[1] Finally, primarily through research on the participants in cardiac rehabilitation (CR) programs, they identified patient characteristics that might hinder successful cardiovascular risk reduction.

Risk Factors and Risk Factor Reduction

Canadians are at high risk of developing CVD. At least one of the following risk factors is characteristic of 8 out of 10 individuals: smoking, physical inactivity, obesity, high blood pressure, diabetes, and dyslipidemia; and 1 in 10 have three or more of these risk factors. The HSFC[2] reported that the prevalence of these risk factors in society may be falsely low because of the nature of the data collection. The findings have been based on the self-reported data of weight, height, and blood pressure. Risk factors can be further subdivided into nonmodifiable and modifiable.[2] In recent years the focus of risk reduction has been further divided into primary prevention of coronary heart disease

(CHD) and secondary prevention strategies aimed at individuals who already have established CHD.

Nonmodifiable Risk Factors

The HSFC[2] defined *nonmodifiable risk factors* as those over which an individual has no control, such as age, gender, ethnicity, family history, and genetic factors. Advancing age is the dominant risk factor for heart disease and stroke because the rates of all major forms of heart disease increase with age. The HSFC[2] noted that the percentage of people who suffer heart problems increases with age. Only 12% of the population in the 50-59 age range report CVD problems, whereas 23% of people in the 70+ age group report problems (p. 53). Stratified by age, the lowest prevalence of at least one risk factor was among subjects ≥ 75 years old (77% of women and 65% of men).[4]

Statistics from the Public Health Agency of Canada suggest that in 1998 the total cost of CVD in Canada was highest after age 35, with 50% of health care costs incurred in the 35-64 age groups and 44.9% in the 65+ age group.[5] Costs for health care were the highest in the 65+ age group for all components of health care costs except long-term and short-term disability[2] (p. 48).

The identification of gender as a nonmodifiable risk factor for CVD has only recently become widely recognized. The gender difference can be seen in the incidence of CVD, mortality rates, and the overall costs of treating CVD. According to data from 2000, gender differences also exist in CVD rates, mortality, hospitalization rates, procedures, and the total cost of care. CVD diagnostic category admission accounts for 21% of all male admissions and 15% of female admissions[2] (p. 36). Admissions for myocardial infarction (MI) are 23% higher for males (p. 39). Mortality rates for both men

and women with CVD continue to decrease, but it remains the leading cause of death (p. 61). The HSFC's[2] findings show that the death rate from MI for men is greater than that for women. In 1999, 36% of all deaths were related to CVDs, with the distribution identical between men and women. According to the HSFC,[2] men die more often from ischemic heart disease and acute MI, whereas women die more often from cerebrovascular accident and congestive heart failure. Deaths caused by CVD increase after age 50 for women, but after age 40 for men. Health Canada's statistics indicate that for women aged 40-49, 14% of deaths are related to CVD; whereas 23% of deaths for men in the same age group are related to CVD.[6] Cardiovascular risk-reduction strategies base treatment intensity recommendations on a clustering of risk factors, one of which is gender.[7]

A family history of early coronary disease is an important risk factor for CVD. The HSFC[2] and Hayman and Hughes[8] identified several components of family history that increase the risk of CVD, including familial factors, lifestyle factors, molecular defects, and genetic vascular physiology. Culleton and Wilson[4] reported that in a case control study in Italy, a family history of MI predicted an increased risk of MI from 1.0 (with no affected relatives) to as high as 20 (with two or more relatives who had MI before age 55). Family history has been evaluated in prospective studies such as the Physicians' Health Study that followed 22,071 men for 13 years and the Women's Health Study that followed 39,876 women for 6.2 years (as cited in Culleton & Wilson[4]). The findings from these two studies indicate that a history of paternal MI at <60 years of age is associated with a greater risk of CVD than infarction is at a later age; in comparison, any maternal history of infarction is associated with a greater risk. Hayman and Hughes[8]

discussed the findings of the Framingham offspring study that indicated that a history of CVD in as least one parent is associated with a significant doubling in risk of CVD for men and a 70% increase in risk for women over an eight-year period. Nasir et al. (as cited in Hayman & Hughes[8]) identified sibling history of CVD as an important factor of family history. They found that a sibling history of CVD may be more strongly associated with subclinical atherosclerosis than parental history of premature CVD. A study of 8,549 asymptomatic individuals found that the odds of having subclinical atherosclerosis increased when both men and women also had a sibling with a history of CVD.[8] These studies support including a family history of CVD as an important risk factor. New research has suggested that other factors such as the presence of elevated levels of homocysteine, C-reactive protein levels, lipoprotein a, and fibrinogen should also be included in an overall CVD risk assessment.[6]

Modifiable Risk Factors

The HSFC[2] defined *modifiable risk factors* for CVD as those over which an individual has some control and that can be modified to reduce the risk of heart disease or stroke. These include smoking, obesity, physical inactivity, diabetes, high blood pressure, and dyslipidemia. The HSFC[2] found that the levels of income and education are consistent predictors in the prevalence of CVD risk factors. Lower-income populations have a higher incidence of all modifiable CVD risk factors, and increased levels of education reduce the prevalence of these risk factors.

Smoking. Hennekens[9] confirmed that smoking increases the risk of developing CVD, and the HSFC[2] found that smoking is a contributing factor in a large proportion of deaths due to CVD. Tanuseputro, Manue, Leung, Nguyen, and Johansen[10] estimated that

smoking is responsible for approximately one quarter of all cardiovascular deaths in Canada. The prevalence of smoking in Canada has decreased approximately 8% from 1985 to 2000-2001; however, 21.7% of the population over the age of 15 years is currently smoking. The highest rate of smokers is in the young and adolescent age group[6] (p. 21). Despite a decrease in prevalence, smoking remains the most dangerous modifiable risk factor in Canada (p. 21). Ambrose and Barua[11] suggested that the adverse effects of cigarette smoking may be maximally activated at relatively low exposures, including secondhand smoke, which elevates the cardiovascular risk among those individuals who have no cardiac history. Studies have failed to show a particular dose-dependent response. Hennekens[9] reported encouraging news that one year after people quit smoking, the risk of MI and death from CVD is reduced by one half, and after several years it begins to approach that of nonsmokers. Data from Wilson, Gibson, Willan, and Cook's[12] meta-analysis suggest that people who quit smoking have a larger reduction in mortality, some as high as 36%. Critchley and Capewell[13] concluded after a two-year follow-up with people who quit smoking that the overall effect of risk reduction occurs quickly.

The major recommendation of the AHA (as cited in Jones, Granger, Short, & Taylor[14]) and the HSFC[2] is that all patients should be encouraged to quit smoking. However, the reduction of smoking is difficult and involves a combination of long-term behavioral support and possibly pharmacologic therapy. The CACR[6] identified strategies to help improve abstinence rates.

Several approaches to behavioral management for smoking cessation have been studied. In their systematic review, Gluckman et al.[15] discussed the effects of individual

counseling on smoking cessation. The 83 patients who received individual counseling for six months or longer had an odds ratio (OR) of 1.62 for successful smoking cessation. Stead and Lancaster[16] also reported consistent evidence that individual counseling increases the probability of cessation compared to less intensive support, and they contended that the evidence is contradictory in support of group therapy as an effective approach compared to advice from a health care professional.

Various pharmaceutical remedies are available with varying efficacy. Silagy, Lancaster, Stead, Mant, and Fowler[17] conducted a meta-analysis of 110 randomized control trials (RCTs) and concluded that all forms of nicotine replacement therapy show evidence of effectiveness (pooled OR = 1.74). Despite the effectiveness of cessation, all methods that utilize only nicotine replacement therapy have a significant relapse rate[6] (p. 178). Pharmacologic therapies, which have a Grade A Level II recommendation, include Buporpion SR, nicotine gum, nicotine inhaler, nicotine nasal spray, and the nicotine patch. Other therapies that are available but have insufficient evidence of their effectiveness[18,19,20,21,22,23] include aversive smoking, interventions to enhance support from the marital partners of those trying to quit, exercise programs, hypnotherapy, acupuncture, acupressure, laser therapy, electrostimulation, naloxone or other opioid antagonist therapy, and anxiolytics. The CACR[6] recommended the addition of pharmacotherapy to behavior therapy and concluded that if smoking cessation is to be effective, an intensive individualized program with or without pharmacological interventions seems to be the most effective, and continued follow-up is essential to decrease the incidence of relapse (p. 177). Studies have shown that combinations of these two strategies can double the abstinence rate.

Obesity. Obesity is another modifiable risk factor. It causes high blood pressure and diabetes, which in turn contribute to the development of CVD. The HSFC[2] defined *excess weight* as a BMI of 25.0-29.9 among individuals aged 18-64 and *obesity* as a BMI of ≥ 30.0 among individuals aged 18-64.

In systematic reviews, Hennekens[9] and Culleton and Wilson[4] discussed their findings from the Framingham Heart Study and the Nurses' Health Study, in which they documented a positive association between body weight and CHD. Data from the Framingham Heart Study, which followed up its participants for up to 44 years, suggested that excess body weight (including overweight and obesity) accounts for approximately 23% of the cases of CHD in men and 15% in women. The Nurses' Health Study considered weight gain after age 18 to 20 years as another determinant of cardiovascular risk. In this study, as an example, the relative risk of a cardiovascular event was 1.2 for a weight gain of 5 7.9 kg after age 18 years, 1.6 for an 8 to 10.9 kg weight gain, and 2.6 for a gain of 20 kg or more. The Framingham study concluded that "it can be estimated that if everyone were at optimal weight, we would have 25 percent less coronary heart disease (CHD), and 35 percent less congestive failure and brain infarctions"[24] (p. 2).

Recent studies have documented dramatic increases in the prevalence of obesity in Canada and the United States, with the rates of obesity nearly tripling over a 15-year period to 15% in 2000. The HSFC[2] found that obesity in men is 1.4 times higher than in women. Rowland[25] argued that the data on reported weight are not as reliable as originally speculated, as evidenced in studies that have demonstrated that the incidence of underreporting actual weight is fairly high. This assertion was further supported in two

studies: Mokdad et al.[26] found that the self-reported prevalence of obesity was 19.8% in the US, whereas Flegal, Carroll, Ogden, and Johnson[27] noted that the measured data for the National Health and Nutrition Examination survey show that the prevalence is as high as 30.5%. There is an alarming increase in the obesity of youth in both countries, which has a significant impact on the future burden of CVD. The epidemic of obesity has been attributed to an overall sedentary lifestyle combined with an overconsumption of calories.

Ardern, Katzmarzyk, Janssen, and Ross[28] and Booth, Gordon, Carlson, and Hamilton[29] found that the distribution of body fat appears to be an important determinant in cardiovascular risk assessment because patients with abdominal (central) obesity are at greatest risk. Both a waist circumference ≥ 100 cm (40 in) in men and ≥ 88 cm (35 in) in women[24] or a high waist-to-hip ratio (WHR) (0.95 in men and 0.85 in women) are associated with an increased morbidity and mortality.[24]

Truncal obesity is associated with a recently identified cluster of CVD risk factors—known as *metabolic syndrome*—that increase an individual's risk of CVD, morbidity, and mortality.[6] Even in the absence of metabolic syndrome, obesity is associated with other emerging and established risk factors for heart disease such as dyslipidemia, pro-inflammatory markers, hypertension, and dysglycaemia.

Weight loss is the main focus of cardiovascular risk reduction in subjects with BMI ≥ 25 kg/m^2. The benefits of weight loss include a reduction in morbidity and mortality, a decreased risk of diabetes mellitus, improved insulin sensitivity, a reduction in CVD, lower blood pressure, lower serum lipid concentrations, and delayed onset and severity of osteoarthritis.[24] These assertions have subsequently been supported by several

studies such as the Swedish Obesity Study, the Nurses' Health Study, and the American National Survey.[24]

Weight reduction will lower serum lipid concentrations and improve glucose tolerance. For example, two-year data on subjects who did or did not lose weight in the Swedish Obesity Study revealed a linear decrease in serum glucose, insulin, and triglyceride concentrations with increasing weight loss.[24] Serum high-density lipoprotein (HDL) cholesterol concentrations rose in parallel with the weight loss. Serum LDL and total cholesterol concentrations do not decrease with weight loss until body weight had decreased by 20%.[24]

Effective interventions for obesity include combining cognitive behavioral therapy techniques with a modest reduction in caloric intake. Weight loss targets should not exceed 0.25-0.5 kg per week. These modest reductions can reduce the risk of CVD by almost 10%.[30]

Diabetes. Adult onset diabetes is a significant risk factor for the development of high blood pressure, stroke, and heart and vascular disease, particularly in women. Diabetes not only increases the incidence of CVDs, but also adversely influences the outcomes.[4] There is a dramatic increase in the prevalence of diabetes with advancing age. In the *Canadian Community Health Survey: A First Look*, Statistics Canada[31] reported that approximately 4.7% of all Canadians have diabetes and that 2%-3% have undiagnosed diabetes and impaired glucose tolerance. Of those affected, 90% have type 2, and the remaining 10% have type 1.[32] Individuals with diabetes have a higher mortality rate from heart disease.[2] The onus has now been placed on health care

professionals to provide intensive identification and aggressive CVD risk-factor modification.

Culleton and Wilson[4] reported that there are conflicting data on the importance of glycemic control in risk reduction and the development of macrovascular disease in patients with type 2 diabetes. In the Prospective Diabetes Study, the United Kingdom Prospective Diabetes Study Research Group[33] found no difference in macrovascular disease between the intensive and conventional therapy groups in the primary analysis. However, a subanalysis suggested that reducing the HbA1c value by 1% was associated with an 18% reduction in MI and a 15% reduction in stroke. This reduction is managed through dietary, lifestyle, and pharmacologic interventions. A discussion of this topic is beyond the scope of this literature review. The National Cholesterol Education Program (NCEP),[7] the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults,[43] and the sixth Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure[34] has recently published guidelines for a framework to treat coronary risk factors aggressively in diabetics and recommended that diabetes be classified as a CHD risk equivalent.[38] The Canadian Cardiac Rehabilitation Society has recommended that all diabetics be treated according to the most recent Canadian Diabetic Association guidelines[6] (p. 138).

Hypertension. The HSFC[2] identified hypertension as one of the major risk factors for CVD. High blood pressure (which the HSFC[2] defined as a systolic blood pressure of ≥140 mmHg or diastolic blood pressure of ≥90 mmHg) increases overall cardiovascular risk by two to three times. Hypertension is a chronic disease that causes pathological

changes to many organ systems as a result of chronically elevated blood pressure. Damage to organ systems leads multiple disease states including MI and stroke[6] (p. 20).

The findings from the *Canadian Community Health Survey*[31] show that the incidence of self-reported hypertension in Canada is 14%. The CACR[6] stressed the importance of increased awareness of hypertension, and Joffres et al.[44] found that 42% of those diagnosed with hypertension were unaware of their condition, 16% were treated and controlled, 23% were treated but not controlled, and 19% were neither treated nor controlled. Goldberg et al.[45] reported that the impact of hypertension in CVD is that 50%-60% of patients who presented with acute coronary had a history of systemic hypertension.

The HSFC[2] consistently found that high blood pressure and its incidence increase with age in a higher percentage of women than men. The assertion that the presence of hypertension is more frequent in women has been challenged by the Canadian Heart Health surveys that have used physician-measured and -diagnosed hypertension figures to show that the prevalence is actually higher in men at 22% than in women at 18%.[44]

Risk-reduction strategies for hypertension include increased awareness, frequent screening, and the use of strategies to reduce overall blood pressure. The 2004 Canadian Hypertension Education Program (CHEP) has recommended that all individuals have their blood pressure checked at every opportunity.[46] Culleton and Wilson,[4] Gluckman et al.,[15] Hennekens and Cannon,[3] Chobanian et al.,[34] and Hennekens[9] supported initiating treatment of patients without CVD or diabetes mellitus if their systolic BP is 140 mmHg or higher or their diastolic BP is 90 mmHg or higher. Randomized trials, including the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial

(ALLHAT), which included 33,357 patients, have demonstrated the clear benefits of using drug therapy to decrease blood pressure and reduce death from MI and CVD. There was no observed difference in the primary combined outcome of fatal CHD or nonfatal MI in patients who took any of the three antihypertensive drugs. Because of their lower cost, thiazide diuretics were considered the preferred first-line antihypertensive agents. Gluckman et al.[15] reported that the findings from several RCTs demonstrate that patients with moderate to severe hypertension or mild hypertension with additional cardiovascular risk factors should be treated with antihypertensive agents. Most of these patients will require two or more drugs.

Pharmacologic agents useful in the control of hypertension and cardiovascular risk reduction include Aspirin, beta blockers, angiotension-converting enzyme inhibitors, angiotension-receptor blockers, thiazide diuretics, and statins. The effectiveness of these agents has been shown in numerous trials.[9,15] Pharmacologic treatment of hypertension needs to be combined with lifestyle changes and health promotion. Pescatello et al.[35] suggested that exercise is the keystone of therapy for the primary prevention, treatment, and control of hypertension. CHEP has suggested an exercise program of 30-45 minutes of moderate dynamic exercise three to five times per week as part of a healthy lifestyle and as an adjunct to pharmacological therapy for hypertension.[36] However, the most effective therapy will control hypertension only if the patient is motivated.[9,34]

Cholesterol. Abnormally elevated cholesterol, LDLs, triglycerides, and low levels of HDLs are important risk factors for the development of coronary artery disease. The lifetime risk of CVD increases sharply with higher total cholesterol levels for men and women of all ages.[37] The HSFC[2] reported that in the 1985-1990 Heart Health Surveys,

45% of men and 43% of women had a total plasma cholesterol level above the desirable level of 5.2 mmol/L and that this level increased with age. They proposed collecting more current data to assess whether progress has been made in decreasing the proportion of the population with high cholesterol levels.

According to the NCEP, LDL lowering should play an important role in primary prevention of CHD; the Canadian Cardiovascular Society supported this recommendation.[38] A 1% reduction in LDL cholesterol reduces the CHD risk by 1%. Recommendations are supported by evidence from multiple animal experimentations, laboratory investigations, epidemiological research, genetic forms of hypercholesterolemia, and controlled clinical trials that indicate a strong relationship between elevated LDL levels and CHD[7] (p. 3200). Secondary prevention trials demonstrate that the reduction of LDL significantly reduces the risk for further coronary events in persons with established CHD (p. 3204). Evidence further shows that using therapy to lower LDL reduces the risk of stroke as well.

The risk reduction of dyslipidemia is complex and involves several strategies, including dietary management, weight reduction, smoking cessation, management of hypertension, and pharmacologic management specifically aimed at reducing LDL and very low density lipoprotein (VLDL) cholesterol levels and increasing HDL cholesterol levels. Target levels for LDL reduction have been refined in the new 2003 guidelines, and the new evidence in the last several years reflects a more aggressive approach to risk management than do the guidelines from 2000.[38]

The primary pharmacologic agents used for cholesterol lowering are the HMG-CoA reductase inhibitors, more commonly known as *statins*. Hennekens[9] reviewed

several trials in which the statin class of drugs was shown to be effective in the primary prevention of CVD. These trials include the Heart Protection Study (6,627 patients), the West of Scotland Coronary Prevention Study (659 patients), and the Anglo Scandinavian Cardiac Outcomes Trial-Lipid Lowering Arm (ASCOT-LLA; 10,305 patients), in all of which several of the statin family drugs were trialed and demonstrated significant mortality and risk reduction. The ASCOT-LLA trial was stopped early because of a statistically extreme benefit to the primary endpoint of MI and fatal coronary artery disease in patients who received atorvastatin. The only contradictory finding came from the Air Force/Texas Coronary Atherosclerosis Prevention Study,[9] in which the researchers found that lipid lowering with pravastatin was not associated with a significant reduction of all-cause mortality or coronary artery disease events. However, they identified and discussed the factors that markedly reduced the statistical power of the study.

Framingham Risk Scoring

The Framingham Heart Study offered a robust method of assessing risk for CHD in the short and long term[7] (p. 3192). This algorithm is based on a series of risk factors. Several cardiovascular societies, including the European, British, Canadian, and American, have adopted the Framingham scoring system, which is used to predict the risk of MI and death. This scoring system is broadly transportable among various populations and assists clinicians in matching risk assessment to intensity of therapy. Framingham risk scoring is not intended to track changes over time, but to provide the clinician with guidelines on the intensity of treatment. At times the Framingham risk

scoring system can provide an underestimation of risk as it does not take into account the presence of other CVD risk factors.[7]

The primary goal of risk assessment is to help define target LDL goals for treatment. The ATP Treatment Group[38] outlines steps to determine LDL goals. The first step is to identify the number of risk factors matched with the 10-year risk of MI and death. Risk is determined initially by scoring the risk factors for the development of coronary artery disease and including scores for age, gender, total cholesterol levels, HDL levels, systolic blood pressure measurements, and history of current smoking. These individuals are then further identified as having CHD and CHD risk equivalents or not. Those with established CHD are at very high risk for developing future events. These individuals can be identified as those who have a history of acute MI or ischemia, unstable or stable angina, and coronary procedures. People can also be classified as having coronary risk equivalents if they have peripheral artery disease, abdominal aortic aneurysms, carotid artery disease (TIA or stroke), or Type 1 or Type 2 diabetes[7] (p. 3229). The ATP III suggests that those with CHD risk equivalents follow the same recommendations for treatment as those with established CHD.

The ATP Treatment Group[38] identified the risk categories as low, moderate, high, and very high. The targets for LDL reduction are LDL <5.0mmol/L for those with a low risk of CHD, LDL <4.0mmolL for those classified as moderate risk, LDL <3.0 for those classified as high risk, and LDL <2.5mmol/L for those classified as very high risk.

Primary and Secondary Prevention

Primary prevention strategies aim to prevent the new onset of CHD. Strategies for primary prevention are supported in an attempt to decrease the burden of heart disease in

the population. It is hoped that preventing disease will reduce the costs and burdens to society. The concept of primary prevention is intended to reduce the risk factors through population and clinically based strategies. Primary prevention is further subdivided into short and long term. The goal of long-term prevention is to reduce the risks of coronary artery disease over the lifespan of individuals who are not at imminent risk of suffering a coronary event, but who have a high probability of developing the disease over their lifespan[7] (p. 3190). Short-term prevention is aimed at individuals who in all probability have advanced atherosclerotic disease but have not yet sustained a coronary event and whose risk of developing one is high (p. 3190). Strategies in primary prevention include assessing an individual's risk of developing CHD and matching them with strategies to reduce the risk.

Secondary prevention strategies are directed toward individuals who have already experienced a coronary event or those who have documented evidence of advanced coronary artery disease. Three major trials were conducted on the use of pharmacological agents in recurrent cardiovascular events. The Scandinavian Survival Study, The Cholesterol and Recurrent Events Study, and the Long-Term Intervention With Pravastatin in Ischemic Disease Study demonstrated reductions in recurrent MI and coronary death, coronary artery procedures, and stroke.[7]

Cardiovascular Risk Factor Reduction Clinics

Reducing cardiovascular risk factors is only as effective as the programs that target the relevant population, particularly with regard to reducing serum cholesterol levels. Cox;[39] Harris, Gipson, and Pearson;[40] Mosca et al.;[41] and Yates, Annis, Pippins, and Walden[42] discussed the elements that hinder successful cardiovascular risk-factor

reduction in the general population. Several elements may limit the effectiveness of current risk-reduction strategies. These researchers identified fragmented care as the greatest threat to quality health care because many patients with CVD are not treated adequately for elevated serum cholesterol and fail to reach the NCEP's recommended cholesterol levels.[43] Despite a national effort to promote measuring cholesterol levels in adults, previous studies have shown that poor control is the norm.

These disparities in care can be addressed by developing specialized risk-reduction clinics. In a systematic review Cox[39] concluded that risk-reduction programs are associated with small but significant changes in CVD risk profiles. DeBusk et al.;[47] Harris et al.;[40] MacLean, Petrasovitis, Connelly, Little, and O'Connor;[48] Mosca et al.;[41] Murchie, Campbell, Ritchie, Simpson, and Thain;[49] and Yates et al.[42] supported Cox's[39] conclusion in studies and reviews and found that formally structured lipid clinics have a significant positive impact on the percentage of patients who reach the NCEP's[43] LDL goals and on the clinics' adherence to the NCEP guidelines for initiating and titrating drug therapy to reduce the overall percentage of LDL. This type of specialized clinic improves the outcomes for patients with and without coronary artery disease. Harris et al.[40] contended that it is not always clear which aspects of a clinic's protocols are important to its effectiveness, and Yates et al.[42] noted that the effectiveness of lipid clinics does not measure how improved medication compliance and lifestyle changes alone might affect these results.

Conceptual Framework for Behavior Change Related to Risk Reduction

The process of cardiac risk-factor reduction is multifaceted, and its success is primarily contingent upon behavioral changes by affected at-risk individuals. Lai and

Cohen[50] pointed out that it is known that lifestyle modification can reduce the risk of coronary artery disease by up to 50%. The AHA (as cited in Jones et al.[14]) advised that "better outcomes can be achieved when exercise is matched with educational efforts promoting risk-factor modification and a healthier lifestyle, and the counseling and support to achieve lasting behavioral change" (p. 2). Basler,[51] Prochaska and Velicer,[1] and Lai and Cohen[50] emphasized the premise that behavioral change needs to be long lasting. Teaching must be based on the principles of adult learning: The information must be relevant, the content and goals must be individualized, feedback must be provided, behavioral goals must be reinforced, and the patient must be guided toward action. Vermeire, Hearnshaw, Van Royen, and Denekens[52] argued that current programming and models based on a paternalistic model should be avoided. The results of current studies, although they have been plagued by imperfect methodologies, consistently demonstrate that a collaborative doctor-patient relationship, communication, and shared decision making are key concepts in predicting patient compliance.

Vermeire et al.[52] and the CACR[6] discussed several theories that explain and predict change and adherence across a range of behaviors. Many of these theories have emerged as prominent in not only predicting adherence, but also providing frameworks to develop therapeutic intervention. A transtheoretical behavioral framework should recognize and aim to facilitate behavior change and promote the self-management of disease, particularly CVD.

The CACR[6] examined several theories for commonalities, including the transtheoretical model of health behavior change, the motivational interviewing theory, the social health-belief model, theories of reasoned action and planned behavior, and the

self-efficacy social cognitive theory. The CACR[6] outlined three important elements that are common among all of these theories related to viewing behavior change as a process that is influenced by multiple factors: individual cognitive processes, variable outcome expectations, and individual self-efficacy perceptions.

Prochaska and Velicer's[1] transtheoretical model of health behavior change has been used to promote effective behavior change. The model outlines several stages of change: (a) precontemplation (no change foreseen within six months), (b) contemplation (change foreseen within six months), (c) action (active behavior change), (d) maintenance (new behavior is practiced until it becomes permanent), (e) relapse (a return to one of the earlier stages), and (f) termination (new pattern of behavior established). Lai and Cohen[50] found that although progress through these stages differs with each person, patients often move into the action stage about one month after a particular educational intervention, then progress to the maintenance stage in three to six months. Progression through the stages is cyclical, and relapse is normal. When patients relapse, they often revert back to the precontemplation or contemplation stage. People also engage in overt or covert activities as they progress through each change stage. These activities include raising consciousness, experiencing dramatic relief, self-reevaluating, environmental reevaluating, developing helping relationships, substituting healthy behaviors, managing contingencies, controlling stimuli, and being socially liberated. Knowledge of these processes is important to practitioners when they plan and implement new strategies to assist patients in dealing with complex behavior change.[6]

The theory of motivational interviewing and social cognitive theory are helpful in guiding the practitioner in planning strategies. The motivational interviewing technique

guides those who seek collaboration with the client rather than confrontation, rely on evocation rather than education, and realize and respect the client's autonomy. The four general principles of this theory are expressing empathy, developing discrepancy from the client's perspective, rolling with resistance from the client rather than confronting it, and supporting self-efficacy behaviors[6] (p. 41). The practical concepts of self-management are drawn from social cognitive theory, which highlights the importance of self-efficacy. The CACR[6] defined *self-efficacy* as "beliefs in one's own capabilities to organize and execute the courses of action required to produce given attainments" (p. 41). Self-efficacy can be promoted by using effective educational techniques and teaching self-management skills such as problem solving, decision making, accessing resources, forming partnerships, taking action, and self-tailoring. These skills are an essential component of the treatment of chronic disease.

Lorig and Holman[53] believed that practitioners involved in programs that promote self-efficacy need to teach their clients the skills required for self-management, and they concluded that programs that focus on the promotion of self-management skills have significantly improved adherence to behaviors such as exercise and relaxation. Specific to CVD is Wheeler, Janz, and Dodge's[54] RCT in which they randomized 452 older women with assorted cardiac diagnosis to a self-managed tailor-made program or to the usual care. They found that the intervention group had 46% fewer inpatient days and a 49% lower inpatient cost.

Vermeire et al.[52] reported that the methodological quality of compliance studies ranges from poor to exceptionally high. Many studies have demonstrated flaws and weaknesses in their design and execution, and the overall design of most of these studies

lacks rigor[6] (p. 43). There is a need for further research that focuses on a description of the illness and clearer definitions of compliance. More research attention needs to be paid to refining assessment approaches and multiple variable analysis and to longitudinal measurement.

Characteristics of Patients Who Achieve Successful Risk Reduction

Behavioral change and risk-reduction strategies must be appropriately planned to address the needs of their target audience. Certain patient characteristics can impact the effectiveness of such programming and, in combination, increase the risks for poor adherence and risk reduction among patients with CVD. Current research on CR has identified certain patient characteristics that can predict poor adherence to behavior change. Patients who were most likely to participate in CR programs were actively referred, educated, and married; revealed high self-efficacy; and had easy access to the rehabilitation programs.[55] Common factors that predict poor adherence to programming include a diagnosis of IHD, the female gender, increased age, and a diagnosis of depression.[55]

Physician endorsement and referral to a risk-reduction program and a positive patient-physician relationship have been identified as the most important predictors of adherence to risk-reduction strategies.[6,61]. Physician attitude is the biggest barrier to successful CR programming, according to the CACR[6] and Gavic.[61] Jackson et al.[55] found that physician endorsement and a positive attitude toward the CR program were the strongest predictors of the number of referrals and ongoing participation in these CR programs in all 12 studies that they examined. Ades, Waldman, McCann, and Weaver[56] concurred with this assertion following their study of over 226 inpatients aged 62 years

and older. They reported that the strength of the primary physician's recommendation and referral was the most powerful predictor of entry into the CR program.

Diagnosis is the most important predictor of the noncompletion of CR programming. Turner, Bethell, Evans, Goddar, and Mullee[57] studied 1,902 patients over a seven-year span and found that patients who had had a previous diagnosis of ischemic heart disease or who had undergone percutaneous angiography with stenting had double the rate of defaulting on the CR programming. Leibowitz, Regess, Manor, Bental, and David[58] found that 91.7% of patients with a diagnosis of ischemic heart disease did not participate in programming, whereas only 56.4% of those with a diagnosis of acute MI did not participate. Jackson et al.[55] disputed this assertion in their systematic review of 10 studies on CR participation rates and found that in 8 of the 10 studies the participation rates were not affected by a primary diagnosis of MI, but were affected by a primary diagnosis of hypercholesterolaemia and PTCA. These patients were also referred more frequently to CR programs.[57] Ades et al.[56] and the CACR[6] suggested that these differences in participation rates may be attributed to the opinion of the severity of the illness and disagreement on the need for treatment.[59] In a study of 143 patients who were aged 65 or older, Petrie, Weinman, Sharpe, and Buckley[60] found that attendance in these programs was significantly related to a stronger belief during admission that the illness could be controlled or cured. Turner et al.[57] identified such weaknesses in the studies as a lack of control groups and incomplete data on the fitness variables, but also saw the number of participants as a strength.

CACR[6] and Gavic[61] found that females are at a higher risk of defaulting on programming and attributed this to several factors that are more prominent among

women. Jackson et al.[55] speculated in their systematic review of over 13 studies that involved 16,804 patients (5,882 of whom were female) that this difference is related not only to the referral of women to CR programming, but also to their participation and adherence rates. Poor adherence factors that cluster in women are increased age, greater comorbidity, higher depression scores, lower initial exercise tolerance, less available social support, and family obligations. The probability of referral to CR programs was lower for three out of four women. They participated less often in CR programs in 13 out of 20 studies that Jackson et al.[55] examined, and married female patients participated less often than married male patients did.

Dunbar-Jacob, Bohachick, Mortimer, Sereika, and Foley[62] examined high-risk populations and found that adherence to medication regimes was higher in the elderly population who had experienced at least one cardiovascular condition. Researchers who conducted studies on CR populations challenged this finding and concluded that age is a major predictor of nonadherence. Elderly people are more likely to default in the completion of programs[57] and are referred less frequently to rehabilitation programs.[61] Leibowitz et al.[58] determined in their study of 439 patients who had been admitted with a CVD diagnosis that the nonparticipation rate in patients greater that age 65 was over 75%.

Grace, Abbey, and Shnek[63] found no significant relation between the effects of depression and the completion of programming. However, Turner et al.,[57] the CACR,[6] and Ades et al.[56] challenged this finding and asserted that depression is a positive predictor of noncompletion of CR programming. They speculated that adherence may be

a mechanism that accounts for the increased mortality and morbidity that result from depression.

The CACR,[6] the HSFC,[2] Jackson et al.,[55] Gavic,[61] and Leibowitz et al.[58] identified other barriers to referral to, participation in, and completion of CR programming, such as long travel times, lower household income, and lower levels of education. The CACR[6] found that adherence to CR programs was higher among those participants who had higher levels of education. Leibowitz et al.[58] discovered that lower education, lower socioeconomic status, and geographic location were also significant barriers to program access and adherence. Barriers that predict poor adherence to behavior change programs in general are frequent daily dosing of medications and treatments, complex dosing regimes, not living alone, multiple disease and polypharmacy, cognitive and/or mental impairment, impaired functional capacity, impaired sensory capacity, low literacy levels, side effects of medications, and trouble swallowing or taking medications. It was noted that $20,000 seems to be the critical level of income that marks the difference between those with good adherence and those with poor adherence.[6,62] These barriers can be extrapolated to other cardiovascular risk populations to determine adherence and successful risk reduction.

Conclusion

The statement that modifiable risk factors for CVD are largely preventable and are mainly a consequence of lifestyle choices has been well documented in the literature. This statement was supported in the literature on the development of clinical practice guidelines and through RCTs, retrospective analysis, and systematic reviews.[2,3] The emphasis in present-day healthcare is on improving risk-factor control, particularly with

strategies to promote behavioral change. Specialized clinics have been promoted to improve adherence and overall risk-factor modification using a variety of approaches. The benefits of these specialty clinics, especially those that focus on lipid reduction, have been extensively identified through various RCTs, retrospective analyses, and systematic reviews. There are gaps in the literature on the characteristics of patients that may predict successful cardiovascular risk reduction. This area requires further research and investigation to develop programming and target it toward those individuals who are not benefiting from current risk-reduction strategies, which in turn can have a positive impact on the overall burden of heart disease on society.

CHAPTER 3

METHODS

Sample

The data used for this study was collected from patients who attended the lipid reduction clinic at the University of Alberta Hospital from 2000 to 2005. Records were received from patients currently followed within this timeframe. Patients had been initially enrolled in the clinic as early as 1994. Of these records 714 cases records were available on the data base that was received from the CRCC. Subjects were then excluded based on information that was missing for calculation of Framingham risk score, baseline LDL levels and follow up LDL levels. Subjects were also excluded that were under the age of 18. A total of 445 cases remained for analysis in this study. A flow chart outlining the cases which were excluded with rationale is presented in Appendix F.

A general description of clinic demographics revealed that between 1991 and 2001 the clinic followed a total of 1,975 patients who were referred from physicians' offices in the Capital Health Region. No formal clinic admission criteria were required for referral. Data were collected on index and follow-up visits to the Cardiovascular Risk Reduction Clinic (CRRC) at the University of Alberta Hospital. Clinical recommendations are forwarded to the referring physician. Each patient is assessed based on information on age, gender, history of hypertension, history of diabetes, smoking history (never, prior, current), the number of pack years, a family history of vascular disease, elevated Lpa levels, elevated homocysteine levels, and history of known vascular conditions (none, previous MI, angina, PTCA, CABG, TIA, Stroke, PVD). The number of risk factors is counted, and a 10-year risk of death from CHD is calculated. A complete

list of current medications is also recorded. Data collected from each follow-up visit include height, weight, BMI, blood pressure and heart rate, waist circumference, hypertension, smoking history, diabetes, family history of vascular disease, transplant, history of known vascular disease, current medications, lipid medications, drug allergies and intolerances, lipid profile date with total cholesterol, LDL, HDL, triglycerides, and TC/HDL ratio recorded with each lab result. Lpa, homocysteine, TSH, fasting glucose, HbA1c, potassium, creatinine, albumin creatinine ration, ALT, AST, CK, CRP, uric acid, and Apo B are documented as dictated by the medical condition. A copy of the data-collection form is included in Appendix A.

Treatments while the patients were enrolled in the clinic involved specified programming aimed primarily at reducing LDL levels and increasing HDL levels. As well, all patients underwent referrals to a dietician, smoking-cessation counselors, Boost Your Heart education programs, consultations with specialists in risk reduction, and healthy-exercise education. Patients were discharged from the clinic once it was determined that they had achieved as much as they could in terms of treatment goals. The number of scheduled visits to the clinic was individually determined, but the number of visits missed was not tracked in the database. Ethics approval was received from the Health Research Ethics Board prior to use of the dataset. The purpose of this cohort analysis was to determine the characteristics of patients who attained target LDL levels measured at their final visit at the cardiovascular risk-reduction clinic at the University of Alberta Hospital.

Objectives

Primary Objective

What are the characteristics of patients who achieve target LDL levels as defined by the Framingham risk scoring system?

Secondary Objectives

1. Do changes in BMI predict the ability to meet target LDL levels?
2. What relationship does the percentage of BMI change have to the percentage of LDL change?

Outcome Variable

The outcome variable was successful risk reduction as measured by the attainment of target LDL levels (defined by the Canadian guidelines[38]) and recommendations for the management and treatment of dyslipidemia. Target LDL levels were calculated on admission to the CRRC. Patients were stratified based on the Framingham risk categories, including (a) low-risk category: LDL <5.0 mmol/L; (b) moderate-risk category: LDL = <4.0 mmol/L; (c) high-risk category: LDL <3.0 mmol/L; and (d) very high risk category: LDL <2.5 mmol/L. Based on the risk categories, the patients were classified as attainment (1) or nonattainment (0) of target LDL.

Predictor Variables

Predictor variables included age, gender, family history of CHD, smoking, BMI on admission, the Framingham risk score based on Canadian guidelines for 2000[38], primary or secondary prevention, number of visits to the clinic, and already within target

LDL levels on admission. The diabetic history was included in the secondary prevention scoring and was therefore not included as an independent risk factor.

Coding of Variables

1. **Age** (measured using an interval scale) was coded as recommended based on the Framingham risk scoring guidelines: <30 years, 35-39 years, 40-44 years, 45-49 years, 50-54 years, 55-59 years, 60-64 years, 65-70 years, and older than 70 years. HSFC[2] states that as age increases so does cardiac risk.
2. **Gender** (measured on nominal scale) was coded as male or female. Gender was included as a predictor variable as HSFC[2] lists gender as a non modifiable risk factor and uses gender to describe populations and outcomes.
3. **Family history** (measured on nominal scale) was coded as *family history* of CHD or *no family history* of CHD. Family history predictor variable was included as HSFC[2] identifies a premature family history of death from a myocardial infarction an important predictor of individual risk.
4. **Smoking history** (measured on nominal scale) as recorded on admission records was *current*, *previous*, or *never*. HSFC[2] identifies smoking as the number one modifiable risk factor of CVD.
5. **BMI** (measured on an interval scale) recorded on admission records was coded as, normal BMI of <25 kg/m^2, overweight BMI as 25-29 kg/m^2, and obese as BMI ≥ 30 kg/m^2. Obesity is identified as a modifiable risk factor by the HSFC[2] and the influence on cardiovascular risk is multifactorial, and example of such is the association between diabetes and obesity[4].

6. **Framingham risk score** (measured on interval scale) was calculated using Canadian Cardiovascular Society guidelines from 2000[38] and was based on scores for gender, age, total cholesterol, HDL cholesterol levels, and systolic blood pressure, combined with additional risk scoring based on the presence of CHD or CHD risk equivalent. The categories were low risk (<10%), moderate risk (10%-20%), high risk (20%-30%), and very high risk (≥ 30%) (Table 1[38]). Framingham risk scoring was used as a predictor variable to help classify and categorize the population and assess the risk levels. The Framingham Heart Study offered a robust method of assessing risk for CHD in the short and long term[7] (p. 3192).
7. **Primary or secondary prevention** (measured on nominal scale) was recorded as *primary prevention* for subjects who had no previous history of coronary artery disease or diabetes and as *secondary prevention* for those with a history of either of the following: myocardial infarction, angina, unstable angina, revascularization procedures, TIA, stroke, or diabetes. Prevention level was used as a predictor as the focus of CVD prevention is to prevent an initial event and it is important to examine the proportion of the population that specialized clinics may be able to assist in preventing a coronary event. Primary prevention is classified as targeting subjects who have not suffered a cardiovascular event as opposed to those who have documented CVD and diabetes[2].
8. **Number of visits** (measured on an interval scale) was coded as the number of follow-up visits to the clinic: 1 for those with only one follow-up visit to the

clinic, 2-4 for those who had at least two visits and no more than four follow-up visits to the clinic, and ≥ 5 as those who were seen more than five times in follow-up. The number of visits to the clinic is useful in identification of individuals who may be at higher risk despite intense interventions.

Table 1

Method for Calculating the 10-Year Risk of Coronary Artery Disease in a Patient Without Diabetes Mellitus or Clinically Evident Cardiovascular Disease Using Framingham Data: 2000 guidelines

Step 1: Determine Risk Points†			**Step 2: Calculate Risk‡**		
	Risk Points			**Risk Points**	
Risk Factor	**Men**	**Women**	**Total Risk Points**	**Men**	**Women**
Age, yrs			1	3	2
30-34	-1	-9	2	4	3
35-39	0	-4	3	5	3
40-44	1	0	4	7	4
45-49	2	3	5	8	4
50-54	3	6	6	10	5
55-59	4	7	7	13	6
60-64	5	8	8	16	7
65-69	6	8	9	20	8
70-74	7	8	10	25	10
Total Cholesterol level, mmol/L			11	31	11
< 4.14	-3	-2	12	37	13
4.15-5.17	0	0	13	45	15
5.18-6.21	1	1	14	≥53	18
6.22-7.24	2	2	15		20
≥ 7.25	3	3	16		24
HDL-C level, mmol/L			17		≥ 27
< 0.90	2	5			
0.91-1.16	1	2	**Step 3: Compare risk with that of average person of same age§**		
1.17-1.29	0	1			
1.30-1.55	0	0	Men		
≥ 1.56	-2	-3	30-34	3	2
Systolic blood pressure, mm Hg			35-39	5	3
< 120	0	-3	40-44	7	4
120-129	0	0	45-49	11	4
130-139	1	1	50-54	14	6
140-159	2	2	55-59	16	7
≥ 160	3	3	60-64	21	9
Smoker			65-69	25	11
No	0	0	70-74	30	14
Yes	2	2	Women		
Record the points			30-34	<1	<1
Age			35-39	<1	<1
Total cholesterol			40-44	2	2
HDL-C			45-49	5	3
Blood Pressure			50-54	8	5
Smoker			55-59	12	7
Add total risk points			60-64	12	8
			65-69	13	8
			70-74	14	8

Note: The Framingham tables underestimate CAD risk if the LDL-C level is ≥ 6.0 mmol/L

‡Risk of CAD outcomes including angina pectoris, unstable angina, nonfatal myocardial infarction and coronary death over subsequent 10 years for a Framingham Study participant with that specific risk score.

§Risk of patient with optimal risk factors

(Fodor et al.,[38] p. 1443)

9. **Within target LDL on admission** (measured on nominal scale) was coded as *At target at baseline* for subjects whose LDL level on admission was already less than or equal to their predetermined target LDL level and *Not at target at baseline* for subjects whose LDL level was greater than predetermined target LDL level. This predictor was included for analysis as it provided a measure of subjects who may have been referred for reasons other than concerns with lipid metabolism disorders not captured by LDL measurement.

For the purposes of secondary analysis, the BMI and LDL were recoded into the following variables:

1. **Obesity class** (measured on nominal scale) on admission was coded as Obese with BMI ≥ 30 kg/m^2 or Not Obese with BMI <30 kg/m^2.
2. **Percent change in BMI** (measured on interval scale) based on changes from admission to last recorded follow-up BMI was coded as no change, ≥ 25% gain in BMI, 0%-25% gain in BMI, 0%-25% loss in BMI, or ≥ 25% loss in BMI.
3. **Percentage change in LDL** (measured on interval scale) based on changes from admission to last recorded follow-up LDL was coded as no change, ≥ 25% increase in LDL, 0%-25% increase in LDL, 0%-25% decrease in LDL, or ≥ 25% decrease in LDL.

Analysis

A retrospective cohort design was used to examine the dataset. Univariate analysis using ANOVA testing was performed to determine significant association between the predictor variables (age, BMI on admission, Framingham risk scoring

criteria [baseline cholesterol, baseline HDL, systolic blood pressure] and number of visits to the clinic) and the attainment of target LDL levels. Chi-square testing was performed to determine significant association between categorical variables (gender, family history of CHD, smoking history, Framingham risk category, level of prevention, and number of subjects already at target LDL on admission) and attainment of target LDL levels.

Logistic regression modeling was performed by entering predictor variables (age, gender, family history, smoking history, BMI, Framingham risk category, prevention, number of visits, base LDL at target on admission) at one time to determine which predictor variables remained independently associated with the attainment of target LDL levels following adjustment. The model was tested for goodness of fit with a Hosmer Lemeshow goodness of fit test.

Secondary analysis was run using descriptive statistics. ANOVA was used to determine significant association between the continuous variable percentage change in BMI and the scale variable of attainment or nonattainment of LDL. Subsequently, these variables were categorized, and Chi-square testing was performed. ANOVA testing was used to determine significant association between the continuous variable percentage change in BMI and the continuous variable percentage change in LDL. These variables were further categorized for Chi-square testing.

CHAPTER 4

RESULTS

For the purposes of this study, 445 cases were included in the analysis. Subjects were enrolled from a minimum of 20 days in the clinic to a maximum of 9 years; the mean time of enrollment was 645 days (21 months). The total number of visits to the clinic ranged from 1 to 11. The mode number of visits was 1, with a mean of 2.39.

Before adjustment, a total of 283 subjects (63.60%) met their predetermined target LDL levels as defined by the Framingham risk scoring system. It is interesting to note that 195 subjects (43.80%) were already within their target LDL levels at admission.

Descriptive Analysis

For a descriptive analysis of the results of this study, see Table 2.

Sociodemographic Variables

1. The *mean age* of the subjects was 40-49 years of age; 54 (12.10%) were under the age of 30, 27 (6.1%) were 35-39, 56 (12.60%) were 40-44, 69 (15.50%) were 45-49, 57 (12.80%) were 50-54, 73 (16.40%) were 55-59, 38 (8.5%) were 60-64, 48 (10.80%) were 65-70, and 23 (5.2%) were 70 or older.
2. 181 (40.7%) of the subjects were *female* and 264 (59.30%) were male.
3. 167 (37.50%) of the subjects denied a family history of coronary artery disease, and 256 (57.5%) reported a family history of coronary artery disease. 22(4.87%) of subjects had no information recorded. This data was confirmed by the clinic staff to include categorize those with a positive family history for a family history of premature death related to CHD as those subjects who had

a father die from a myocardial infarction before age 55 or a mother who died from a myocardial infarction before age 65.

4. 212 (47.60%) of the subjects never smoked, 157 (35.30%) of the subjects previously smoked, and 76 (17.10%) of the subjects were current smokers. This data was self reported by the subjects.

Table 2

Frequency Table for Independent and Dependent Variables

Variable	Frequency	Percentage
Age 10-year category		
<30 yrs.	54	12.10
35-39 yrs.	27	6.10
40-44 yrs.	56	12.60
45-49 yrs.	69	15.50
50-54 yrs.	57	12.80
55-59 yrs.	73	16.40
60-64 yrs.	38	8.50
65-69 yrs.	48	10.80
≥ 70 yrs.	23	5.20
Gender		
Female	181	40.70
Male	264	59.30
Family HX		
No	167	37.50
Yes	256	57.50
Missing: System	22	4.87
Smoking		
Never	212	47.60
Previous	157	35.30
Current	76	17.10
BMI recoded		
<25 normal	75	16.90
25-29 obese	157	35.30

Table continues on next page

Variable	Frequency	Percentage
≥ 30 morbidly obese	153	34.40
Missing: System	61	13.50
FCA with 2nd		
Valid: Low risk	187	42.00
Moderate risk	101	22.70
High risk	40	9.00
Very high risk	117	26.30
Base TC recoded		
Valid: <4.14	34	7.60
4.15-5.17	61	13.70
5.18-6.21	125	28.10
6.22-7.24	93	20.90
≥ 7.25	132	29.70
Base HDL recoded		
Valid: <0.9	88	19.80
.91-1.16	143	32.10
1.17-1.29	62	13.90
1.30-1.55	86	19.30
≥ 1.56	66	14.80
Systolic BP		
<120	83	18.70
120-129	87	19.60
130-139	88	19.80
140-159	121	27.20
≥ 160	66	14.80
Total	445	100.00
Prevention		
Primary	337	75.70
Secondary	108	24.30
Visits recoded		
1 FU visit	203	45.60
2-4 FU visits	180	40.40
≥ 5 FU visits	62	13.90
Met LDL target		
Did not meet LDL target	161	36.40

(table continues)

Variable	Frequency	Percentage
Met LDL target	283	63.60
Base at target		
Not at target at baseline	250	56.20
At target at baseline	195	43.80
BMI		
Obese: ≥ 30 kg/m^2	153	34.40
Not obese: <30 kg/m^2	232	52.10
Missing	145	
Percentage change in BMI		
No change	8	1.80
≥ 25% gain	4	0.90
0-25% gain	162	36.40
0-25% loss	120	27.00
≥ loss	6	1.30
Missing: System	145	32.60
Weight gain or loss		
No weight change	8	1.80
Weight gain	166	37.30
Weight loss	126	28.30

Clinical Variables

1. On admission, 75 (16.90%) of subjects had a recorded BMI of <25 kg/m^2, 157 (35.30%) were classified as overweight with a BMI of 25-29 kg/m^2, and 153 (34.40%) were classified as obese with a BMI of ≥ 30 kg/m^2. 145 (32.60%) had missing data regarding BMI.
   - 153 (34.4%) of the subjects were classified as obese with a BMI ≥ 30 kg/m^2.
   - 8 (1.8%) of the subjects had not change at all in their BMI, 4 (0.9%) had a ≥ 25% gain in BMI, 162 (36.4%) had a gain of up to 25%, 120 (27%) had

lost up to 25% of their initial BMI, and 6 (1.3%) lost ≥ 25% of the admission BMI.

- Overall 166 (37.3%) of the subjects gained weight, 126 (28.3%) lost weight, and 8 (1.8%) had no change in their weight.

2. 187 (42.00%) of the subjects were classified according to the Framingham risk category as low risk (<10%) for death from CAD (target LDL <5.0mmol/L), 101 (22.70 %) as moderate risk (10%-20%; target LDL <4.0mmol/L), 40 (9.00%) as high risk (20%-30%; target LDL <3.0mmol/L), and 117 (26.30%) as very high risk (≥ 30%; target LDL <2.5).
   - 34 (7.6%) of the subjects' total cholesterol values fell below 4.14mmol/l, 61 (13.7 %) ranged from 4.15-5.17mmol/L, 125 (28.1%) ranged from 5.18-6.21mmol/L, 93 (20.9%) ranged from 6.22-7.24mmol/L, and 132 (29.7 %) were above 7.25mmol/L.
   - 88 (19.80%) of the subjects had HDL levels below 0.9, 143 (32.10%) ranged from 0.91-1.16, 62 (13.90%) ranged from 1.17-1.29, 86 (19.30%) ranged from 1.30-1.55, and 66 (14.80%) were greater than 1.56.
   - 83 (18.7%) of the subjects had a systolic BP <120mmHg, 87 (19.6%) ranged from 120-129mmHg, 88 (19.8%) ranged from 130-139mmHg, 121 (27.2%) ranged from 140-159mmHg, and the remaining 66 (14.8%), ≥ 160mmHg.
   - Following the addition of scores for the 10-year risk of CAD, further subjects were then placed in Framingham risk categories based on a history of coronary heart disease or a coronary heart disease risk

equivalent (refer to Method section). Of these subjects, 49 (11%) reported a history of previous myocardial infarction, 37 (8.3%) reported a previous history of angina, 29 (6.5%) reported a history of PTCA procedures, 38 (8.5%) reported having undergone coronary bypass surgery, 11 (2.5%) reported a previous history of TIA, 9 (2%) reported a history of stroke, and 96 (21.6%) reported no history of vascular disease.

3. 337 (75.70%) of the subjects were being followed for primary prevention, and the remaining 108 (24.30%) for secondary prevention.
4. 203 (45.60%) of the subjects had one follow-up visit to the clinic, 180 (40.40%) had 204 follow-up visits, and 62 (13.90%) had ≥ 5 follow-up visits.

Objective 1

What are the characteristics of patients who achieve target LDL levels as defined by the Framingham risk scoring system?

Hypothesis 1

There will be no significant difference among the independent predictor variables (age, gender, family history, smoking history, BMI, Framingham risk category, prevention, number of visits, base LDL at target on admission) and the attainment of target LDL levels ($p \geq 0.05$).

Univariate analysis. Univariate analysis was used to compare the independent predictor variables (age, gender, family history, smoking history, BMI, Framingham risk category, prevention, number of visits, base LDL at target on admission), including those required to calculate the Framingham risk category (total cholesterol, HDL cholesterol,

and systolic BP) with the attainment of target LDL. The independent predictors with significant association ($p < 0.05$) are described first.

ANOVA testing (Table 3) indicated that age was a significant ($p < 0.001$) predictor of the ability to meet target LDL levels. Subjects who met target LDL were younger (mean age = 49.4 years) than those who did not meet the target LDL levels (mean age = 54.12 years). BMI was a significant ($p = 0.04$) predictor of the ability to attain the target LDL. Subjects who met the target LDL levels had a greater mean BMI (32.41 kg/m^2) than did those who did not meet their target LDL levels (mean BMI = 28.33 kg/m^2). The number of visits to the clinic was found to be a significant ($p = 0.001$) predictor of the ability to meet target LDL levels. Subjects who attained the target LDL had fewer mean visits (2.17) compared to those who did not attain the target LDL (mean visits = 2.77).

Table 3

ANOVA Table of Comparison of Means to Meet Target LDL

	Did not meet target LDL		Met target LDL		
Variables	N	Mean	N	Mean	P value
Age	162	54.21	283	49.12	0.00
BMI admission	162	28.33	283	32.41	0.04
Number of visits	162	2.77	283	2.17	0.00

Categorical data were analyzed using the Chi-square method (Table 4). Analysis indicated that those subjects with no prior smoking history were significantly ($p = 0.001$) more likely to attain their target LDL levels. Subjects who had never smoked were more likely to reach their target LDL levels (53.7%) than were those who had previously smoked (30.0%) and those who were current smokers (16.3%). Analysis indicated that

those subjects classified as low risk by Framingham scoring were significantly ($p = 0.001$) more likely to attain their target LDL levels than were the subjects classified in the other categories. Subjects classified as low risk were more likely to reach their target LDL levels (56.5%) than were those categorized moderate risk (21.2%), high risk (6.0%) and very high risk (16.3%). There was a significant difference ($p = 0.001$) between individuals who were being followed for primary or secondary prevention and

Table 4

Crosstab Comparison Variables With Met Target LDL

Category	Did not meet LDL	Met target LDL	P value (Chi sq.)
Gender			
Female	40.7%	40.6%	0.98
Male	59.3%	59.4%	
Family history			
Yes	57.1%	62.5%	0.17
No	42.9%	37.5%	
Smoking history			
Never	37.0%	53.7%	0.00
Previous	44.4%	30.0%	
Current	18.5%	16.3%	
Framingham category			
Low risk	16.7%	56.5%	0.00
Moderate risk	25.3%	21.2%	
High risk	14.2%	6.0%	
Very high risk	43.8%	16.3%	
Prevention			
Primary	59.9%	84.8%	0.00
Secondary	40.1%	15.2%	
Base LDL at target			

Table continues on next page

Category	Did not meet LDL	Met target LDL	P value (Chi sq.)
Not at base	86.4%	38.9%	0.00
At target at base	13.6%	61.1%	

those who attained their target LDL levels. Subjects who were being followed for primary prevention were more likely to reach their target LDL levels (84.8%) than were those who were followed for secondary prevention (15.2%). Already being within the target LDL level was a significant ($p = 0.001$) predictor of the ability to attain target LDL levels at follow-up. Subjects whose LDL levels were already at target on admission were more likely to meet target LDL levels (61.1%) than were those whose LDL was not within target range on admission (38.9%) (Table 3). Categorical testing indicated that the variable gender and variable family history of CHD did not significantly predict the attainment of target LDL levels.

Logistic regression. Logistic regression modeling was used to test the relationship among the variables and the attainment of target LDL levels. The results of the modeling (Table 5) indicate that gender, a BMI ≥ 30 kg/m^2, a low-risk Framingham, the number of visits, and already being at target LDL on admission were significant independent predictors of attaining target LDL levels. Males (odds ratio = 2.01, $p = .02$, male compared to females), those with a BMI ≥ 30 kg/m^2 (odds ratio = 2.81,$p = 0.01$, compared to those with BMI <25 kg/m^2); those with a low risk of CHD (odds ratio = 6.69, $p = .04$, compared to those at very high risk); those who attended one follow-up at the clinic (odds ratio = 2.33, $p = 0.03$, compared to those who had more than five follow-up visits); and those who were already at baseline LDL levels on admission (odds ratio = 5.16,$p = 0.00$, compared to those who were not within their target LDL on admission) were more likely to meet their predetermined target LDL levels. The odds

ratio for the variables of age, smoking history, family history, and prevention type were non significant predictors of success. The Hosmer Lemeshow test indicated a goodness of fit of the model at p = .735.

Table 5

Logistic Regression Table: Independent Variables to Meet LDL Target

			95% confidence intervals	
Variables	POR	P value	Lower	Upper
Age				
<40 yrs.	1.00			
40-50 yrs.	1.41	0.47	0.56	3.55
50-60 yrs.	1.89	0.20	0.71	5.01
≥ 60 yrs.	1.32	0.60	0.46	3.77
Gender				
Female	1.00	0.02	1.10	3.68
Male	2.01			
Family history				
None	1.00			
Present	1.02	0.94	0.59	1.76
Smoking history				
Current	1.00			
Never	1.68	0.19	0.77	3.62
Previous	0.77	0.51	0.35	1.69
BMI admission				
<25 normal	1.00			
25-29 overweight	1.67	0.16	0.82	3.47
≥ 30 obese	2.81	0.01	1.34	5.88
Framingham risk				
Very high	1.00			
Low	6.69	0.04	1.15	40.12
Moderate	2.00	0.41	0.38	10.45
High	1.35	0.73	0.24	7.61
Prevention type				
Primary	1.00			
Secondary	1.23	0.81	0.24	6.26
Number of visits				
5 follow-up	1.00			

Table continues on next page

1 follow-up	2.33	0.03	1.02	4.83
2-5 follow-up	1.93	0.10	0.83	4.02
Base LDL on admission				
Base LDL not at target	1.00			
Base LDL within target	5.16	0.00	2.71	9.82

Objective 2

Do changes in BMI predict the ability to meet target LDL levels?

Hypothesis 2

There will be no significant difference between changes in BMI and the attainment of dependent variable target LDL levels ($p \geq 0.05$). The results of ANOVA testing indicated no significant relationship between changes in BMI and attainment of target LDL levels ($p \geq 0.05$).

Objective 3

What is the relationship between percentage changes in BMI and percentage changes in LDL?

Hypothesis 3

There will be no significant relationship between the percentage of change in BMI and the percentage of change in LDL ($p \geq 0.05$). ANOVA testing was performed using continuous variables, and the results indicate no significant relationship. Subsequently, these variables were categorized, and the Chi-square results indicate that no significant relationship existed.

Primary and Secondary Prevention (Subgroup Analysis)

Any discussion on the findings of a study that is focused on successful risk reduction would not be complete without an analysis of the ability to achieve the ultimate targets of risk reduction: prevention of myocardial infarction, prevention of coronary heart disease requiring revascularization procedures, and prevention of cerebrovascular events. Primary prevention strategies are aimed at reducing the development of these conditions, whereas secondary prevention strategies are directed towards individuals who have already experienced a coronary event or those who have documented evidence of advanced coronary artery disease. Three major trials (the Scandinavian Survival Study, the Cholesterol and Recurrent Events Study, and the Long-Term Intervention With Pravastatin in Ischemic Disease Study) demonstrated reductions in recurrent MI and coronary death, coronary artery procedures, and stroke with secondary prevention strategies.[7] The dataset contained valuable information on the ability of the clinic to achieve "absolute" risk reduction.

On admission, 376 subjects reported no previous history of vascular events, and 69 reported a previous history (myocardial infarction, coronary artery procedures, stroke, and TIA). Chi-square testing showed a significant relationship ($p = 0.001$) between subjects with a history of previous vascular events and those with a history of recurrent vascular events. Of the 69 subjects with a previous history of vascular events, 84.1% reported a recurrent vascular event during follow-up and 15.9% of the individuals with no previous history of vascular events reported at least one vascular event at follow-up (Tables 6 and 7).

Table 6

ANOVA Table of Comparison of Means to Follow Up Vascular Events

	No vascular event on follow-up		Vascular event on follow-up		
					Table continued on next page
Variables	N	Mean	N	Mean	P value
Age	376	49.79	69	57.41	0.001
BMI admission	321	30.76	64	32.17	0.58
Number of visits	376	2.26	69	3.12	0.001

Table 7

Crosstab Comparison: Predictor Variables and Incidence of Vascular events

Predictor variables	No vascular event on follow-up	Vascular event on follow-up	P value (Chi sq.)
Gender			
Female	42.6%	30.4%	0.06
Male	57.4%	69.6%	
Smoking history			
Never	49.7%	36.2%	0.051
Previous	33.0%	47.8%	
Current	17.3%	15.9%	
Framingham category			
Low risk	48.1%	8.7%	0.001
Moderate risk	26.3%	2.9%	
High risk	10.4%	1.4%	
Very high risk	15.2%	87.0%	
Prevention			
Primary	86.7%	15.9%	0.001
Secondary	13.3%	84.1%	

(table continues)

Predictor variables	No vascular event on follow-up	Vascular event on follow-up	P value (Chi sq.)
Family history			
Yes	39.6%	39.1%	0.94
No	60.4%	60.9%	

CHAPTER 5

DISCUSSION

The statement that modifiable risk factors for CVD are largely preventable and are mainly a consequence of lifestyle choices has been well documented in the literature.[2,3] The emphasis in present-day healthcare is on improving risk-factor control, particularly with strategies to promote behavioral change. Specialized clinics have been developed to improve adherence and overall risk-factor modification using a variety of approaches. The 'expected' benefits of these specialty clinics, especially those that focus on lipid reduction, have been amply reported in the literature. In contrast, the characteristics of patients that may predict successful cardiovascular risk reduction was identified as an area that requires further research and investigation.

The purpose of this research study was to determine the patient characteristics that predict successful cardiovascular risk reduction (as defined by the attainment of predetermined target LDL levels) and, furthermore, to look specifically at the association between changes in weight and changes in LDL levels. The data for this study were obtained from the CRRC at the University of Alberta Hospital in Edmonton, Alberta. The results of the primary analysis provide insight into the characteristics that may predict successful outcomes.

The results of this study suggest that patients who were followed at the CCRC were generally successful at achieving risk reduction as measured by the attainment of target LDL levels. Of the subjects in the sample, 63.6% met their target LDL levels at their final follow-up visit. However, it is interesting note that 43.8% of the subjects were already within target LDL levels at their index clinic visit. Furthermore, after controlling

for patients' sociodemographic and clinical characteristics, being at target LDL at the index clinic visit was independently and significantly predictive of the ability to attain follow-up target LDL levels. The reasons that may explain why such a large portion of the study subjects were already at target LDL levels on admission to the CRRC are multifactorial. These factors may have included referral to the clinic, the presence of other emerging risk factors, physician referral bias, the demographic region served, and accessibility to the clinic and contributed to the fact that some patients were at target LDL on admission. One noteworthy finding of this analysis is that of the 66.6% of the patients who did not meet target LDL at follow-up, 11.3% of those who were already within the baseline LDL on admission **did not remain** within target range at follow-up, and 56% of the individuals who were not at baseline on admission failed to meet baseline target LDL levels.

Logistic modeling of the data indicates four additional independent predictors of success: **gender**, a Framingham classification of **low risk** of heart disease compared to a very high risk, one **follow-up visit** compared to ≥ 5 , and a **BMI of** ≥ 30kg/m^2 compared to <25kg/m^2.

Gender

Gender was found to be an independent predictor of outcome once other factors were controlled for. **Males** were significantly more likely to achieve success than were females. This is similar to the findings from research on cardiac rehabilitation. CACR[6] and Gavic[61] found that females are at a higher risk of defaulting on programming. Jackson et al.[55] speculated that clusters of poor adherence factors in women included increased age, greater comorbidity, higher depression scores, lower initial exercise

tolerance, less available social support, and family obligations. Factors that may limit the success of women in reducing risks may be related to lifestyle, including preferences for exercise modalities; family demands on personal time; the need to cook for several family members with various dietary requirements; the availability of transportation, funds, and a social support network; and encouragement from significant others and family. This finding indicates that clinicians and program planners need to revaluate service delivery methods for women and that providing gender-specific programming may increase the success of the programs.

Framingham Risk Category

Individuals who were classified as at low risk of CHD were significantly more likely to achieve success compared to those in the very high risk **Framingham category**. This finding is intuitively consistent based on the Framingham classification system. We know that to be classified as in the lowest Framingham category, which indicates a low risk of death from cardiovascular disease, the presence of other recognized risk factors must be lower than in those individuals classified in the very high risk category. Therefore, as the risk level increases, the overall targets for risk reduction are stricter, and the goals become more difficult to achieve. At times the Framingham risk scoring system can provide an underestimation of risk as it does not take into account the presence of other CVD risk factors. [7]A significant association was found between a low risk and the LDL levels within target on admission. In the low-risk category, 71.3% of the subjects were already at target LDL on admission. Caution must be noted when interpreting data among subjects who did not achieve their target LDL levels that were classified in the higher risk

categories. Some of these subjects were referred to the clinic only after exhaustive measures to reduce their LDL levels had been attempted by the referring physician.

Gender is significantly associated with risk level, which is evident in the fact that 55% of the females compared to 33% of the males were classified as low risk. Only 15.5% of the females were classified as very high risk, compared to 33.7% of the males. Why did fewer females achieve successful risk reduction when so many of them were at lower risk on admission? There are several clinical implications to this finding. The literature recommended that programming be delivered in gender-specific models. The factors that influence how well women at low risk adhere to therapeutic interventions may include their perceptions of illness severity, pressure from family demands, social support, side effects of medications, and their perceptions of being caregivers. These factors need to be considered in planning care specifically for female patients.

Number of Follow-Up Visits

The subjects who were assessed once in **follow-up** were significantly more likely to meet target LDL levels than were those who required more that five visits to the clinic. A total of 46.6% of the subjects had one follow-up visit, 40.4% had two to four, and 13.9% required more than five. Individuals who were seen only once in follow-up most likely required an initial assessment for risk stratification, an evaluation for risk factors, advice on lifestyle modifications, and perhaps some minor medication changes, followed by a visit to assess the effectiveness of the recommendations. Risk factor reduction care was transferred back to the referring physician when clinic staff determined that the interventions resulted in maximum benefit. Change theory may offer some explanation for the variability in the number of clinic visits in this sample. Lai and Cohen[50] found that

although progress through various stages of change differs with each person, patients often move into the action stage about one month after a particular educational intervention, then progress to the maintenance stage in three to six months. Progression through the stages is cyclical, and relapse is normal. Understanding behavior change theory may assist in determining individual progress through the stages of change and evaluating individual programming to ensure that the interventions are also individualized. It is reasonable to expect that as the stages of change become more challenging and complex, the progress will be slower.

Obesity

Individuals with a **BMI** ≥ 30 kg/m^2 at the initial clinic visit were significantly more likely to meet their target LDL levels. On admission, 16.90% of the subjects had a BMI of <25 kg/m^2; 35.30% were classified as overweight, with a BMI of 25-29 kg/m^2; and 34.40% were classified as obese, with a BMI of ≥ 30 kg/m^2. Even though the distribution of the subjects in the latter two categories was similar, only the highest BMI category demonstrated a significant association with the achievement of target LDL levels. Research that focused on obesity reduction supported weight loss as the main focus of cardiovascular risk reduction in individuals whose BMI is ≥ 25 kg/m2. The findings of a systematic review of several obesity studies reveal that serum LDL and total cholesterol concentrations do not decrease with weight loss until body weight has decreased by 20%.[24] Bray[24] discussed the benefits of weight loss, including reduced cardiovascular disease, lower blood pressure, and lower serum lipid concentrations.[24] Several studies, such as the Swedish Obesity Study, the Nurses Health Study, and the American National Survey[24] have subsequently supported these assertions. There was no

significant difference found with ANOVA testing between the percentage of BMI change and success in attaining target LDL levels. No significant association was found when the percentage of BMI change was compared categorically with the attainment of LDL targets levels. Furthermore, no significant association was found when the percentage of BMI change was compared with the percentage of LDL reduction. These finding are interesting when we evaluate the effectiveness of our weight-reduction programming. Research and clinical practice have supported the belief that people manage better with other risk factors when weight is reduced. Workload on the heart is reduced, exercise tolerance is increased, nutritional intake is improved, and individuals tend to have a greater sense of well being.

Primary and Secondary Prevention

Although the findings of this dataset may not be applied to general populations, the results indicate that perhaps service delivery models should be revaluated. As previously mentioned, research on cardiovascular risk reduction has been conducted primarily in controlled settings. This study provides valuable insights into the effectiveness of such strategies in uncontrolled settings, because the rate of recurrent coronary events in the secondary prevention population is concerning. The predictor variables that are significantly associated ($p < 0.05$) with recurrent vascular events include age, number of visits, Framingham risk category, and prevention. Do additional factors contribute to repeat event rates that are not captured in this study? Future research would be beneficial to study the factors that affect the secondary prevention population and the recurrence of events.

Clinical Implications

Clearly, risk-reduction strategies are a valuable and important resource. Strategies that the CRRC at the University of Alberta Hospital uses and strategies for risk reduction in general include smoking-cessation counseling, weight-management programs that involve counseling by dieticians, and cholesterol medication management counseling. This dataset presented a good opportunity to address risk reduction in an established program. The overall effectiveness of these strategies can be evaluated by examining the outcomes of obesity management and smoking cessation.

Obesity Reduction

On admission, 35.30% of the subjects were classified as overweight, with a BMI of 25-29 kg/m^2; and 34.40% were classified as obese, with a BMI of ≥ 30 kg/m^2. Subsequently, 1.8% of the subjects had no change in their BMI, 0.9% had an increase of $\geq 25\%$, and 36.4% had an increase of up to 25%. Furthermore, 27% of the subjects had a decrease in their BMI of up to 25%, and 1.3% had a decrease $\geq 25\%$. Overall, 37.3% of the subjects gained weight, 28.3% lost weight, and 1.8% had no change in their weight. Categorical testing indicated a significant association ($p = 0.039$) between BMI class and percentage of weight change. If a primary goal of nutritional counseling is helping individuals to reduce weight, the question arises, What factors might be responsible for the large proportion of individuals who actually gained weight while enrolled in the program? We know that behavior change is difficult and that individual progress through each stage of change is different. We cannot control for individual motivation patterns, access to adequate nutritional choices, and access and motivation to exercise. This finding requires the evaluation of service delivery methods. Specifically, more

information is required to determine whether there is a more effective way to target programming to those who tend to gain weight, such as ensuring access in the community to gender- and age-specific programming.

Smoking Cessation

The major recommendation of the AHA (as cited in Jones et al.[14]) and the HSFC[2] is that all patients be encouraged to quit smoking. However, the reduction of smoking is difficult and involves a combination of long-term behavioral support and possibly pharmacologic therapy. The CACR[6] recommended the addition of pharmacotherapy to behavior therapy and concluded that if smoking cessation is to be effective, an intensive individualized program with or without pharmacological interventions seems to be the most effective, and continued follow-up is essential to decrease the incidence of relapse (p. 177). Studies have shown that combinations of these two strategies can double the abstinence rate.

Patients who smoke are counseled on smoking cessation by certified smoking cessation counselors while enrolled in the CRCC. Categorical testing of the data showed significant association ($p = 0.001$) between the subjects' smoking history and the follow-up smoking history. While they were followed at the clinic, 1.4% of the subjects who had never smoked started to smoke, 2.5% of the subjects who had previously smoked started again, 61.8% of the subjects who were current smokers continued to do so, and 23.7% quit smoking. These findings that demonstrate low success with smoking cessation concur with the findings in the literature on the overall difficulty of achieving success. Smoking-cessation programming requires the constant evaluation of service delivery models and the adjustment of strategies to achieve a higher rate of success.

Limitations

The limitations of this study are inherent in using a secondary dataset. First, it is assumed that all subjects were exposed to similar treatment programs and that all had the same opportunity to participate in the clinic. Secondly, some of the subjects may have been referred to the clinic for variables that are not captured in this current database, and the level of LDL may not have been the primary reason for referral. Thirdly, no program can control all facets of the participants' progress through the health continuum, such as the individual physiological disease process, their motivation to visit the clinics, the availability of transportation to the clinic, their ability to afford medications or adhere to prescribed exercise regimes, or the number of at-risk individuals actually referred to the clinic by their primary practitioners. Additional assumptions are made about the consistency of the data collection, the techniques used to collect the data, the tests for reliability, and the consistency of the equipment used for data collection. The influence of comorbid conditions was not controlled for, and utilizing a nonexperimental retrospective correlational design helped to limit this effect. The findings can be generalized only to the target population.

Originally, a sample of data from 714 subjects was reviewed. Subjects with missing data that was required to calculate the Framingham risk score were excluded. Analysis of the missing data found no significant association ($p < 0.05$) between the 269 subjects who were excluded because of missing data that were required to calculate the Framingham scores and those who had all of the data required for the Framingham calculations. Some of the excluded subjects had missing baseline LDL levels that could be related to higher than average triglyceride levels and would affect the calculation of

total cholesterol levels. It would be interesting to examine these variables in future research. The categorizing of patients as successful based on attaining target LDL levels is limited by the collection of the data, as this level was collected at a static point of time and may not necessarily be reflective of changes that may have occurred after this time.

Research Implications

This rich dataset leads to a number of future research questions: What are the characteristics of patients who achieve target HDL levels? Are there associations between reduced HDL levels and the attainment of target LDL levels? What are the characteristics of patients who meet target cholesterol levels? Is there a relationship between total cholesterol level reduction and recurrent vascular events? Examining all of these questions would help to evaluate the effectiveness of risk-reduction strategies outside of the controlled setting.

CHAPTER 6

CONCLUSION

The burden of heart disease on patients and society has been well documented, and the investigation and treatment of risk factors for cardiac disease have been identified as important components of cardiovascular care. The literature discussed the prevalence of risk factors and their influence on CVD mortality, emphasized the need for specialty clinics to manage risk factors, and demonstrated the effectiveness of specialized programming in improving CVD mortality. Gaps in the literature exist with regard to a description of patients who successfully reduce their risk of mortality from cardiovascular disease through participation in a risk-reduction program. With this information, strategies can be developed to improve the risk reduction in the population who are not currently attaining their maximum potential.

The purpose of this study was to identify patient characteristics that independently predict successful cardiovascular risk reduction. Success was measured as the attainment of target LDL levels that were determined upon admission to the CRRC. The dataset used for this study was collected from patients who attended the CRRC at the University of Alberta Hospital between 2000 and 2005, and a retrospective cohort design was used to examine the dataset. A variety of statistical methods were used to test association and predict relationships between predictor variables (gender, smoking history, Framingham risk category, family history, number of visits, prevention, base LDL on admission already at target, age, BMI). Logistic regression modeling was performed to determine which predictor variables were independently associated with attaining target LDL levels following adjustment. Secondary analysis was conducted using descriptive statistics to

determine significant association between a change in BMI and the scale attainment of LDL and the percentage change in LDL.

The results of logistic regression modeling indicated that gender, a low-risk Framingham, a BMI of ≥ 30 kg/m^2, the number of visits, and already being at target LDL on admission were significant independent predictors of the ability to attain target LDL levels. Secondary analysis resulted in no significant association between the percentage of BMI change and the attainment of target LDL levels or the percentage of reduction in LDL. Overall, 37.3% of the subjects gained weight, 28.3% lost weight, and 1.8% had no change in their weight. Categorical testing indicated a significant association ($p = 0.039$) between BMI class and percentage of weight change.

Discussion of the findings of a study focused on successful risk reduction would not be complete without an analysis of the ability to achieve the ultimate target of risk reduction, the prevention of myocardial infarction and an examination of the need for revascularization procedures and cerebrovascular events. In the study 84.1% of the 69 subjects with a previous history of vascular events reported a recurrent vascular event during follow-up, and 15.9% of those with no previous history of vascular events reported at least one vascular event at follow-up.

The overall effectiveness of risk-reduction strategies can be evaluated by examining the outcomes of obesity management, smoking cessation, and medication management programs. While they were being followed at the clinic, 1.4% of the subjects who had never smoked started to smoke, 2.5% of those who had previously smoked started again, 61.8% of those who were current smokers continued to do so, and 23.7% quit smoking.

Limitations in studies are inherent with using a secondary dataset. An analysis of missing data found no significant association between the 269 subjects excluded because of missing data required for the calculation of Framingham scores and those with all of the data required for Framingham calculations. Future research using this dataset could explore the relationships between other variables such as HDL and total cholesterol. Examining all of these questions would help to evaluate the effectiveness of risk-reduction strategies outside of the controlled setting.

In summary, it is clear that although the research identified the importance of and supported the use of risk-factor reduction strategies to reduce the burden of cardiovascular disease, evidence is scarce to support the effectiveness of strategies in meeting target objectives outside of the controlled setting. The answers found in this study can help researchers to identify the characteristics of patients who may benefit from current programming and alter service-delivery models to benefit those who are not meeting target LDL levels. Further research is needed to examine the characteristics of patients from other populations who are in risk-reduction programs to assess whether these findings remain consistent with the general population. If future research identifies similar trends, then programs will need to be altered to improve the outcomes.

REFERENCES

1. Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*(1), 38-47.

2. Heart and Stroke Foundation of Canada. (2003). *The growing burden of heart disease and stroke in Canada*. Ottawa, ON: Author.

3. Hennekens, C. H., & Cannon, C. (2004). *Risk factor reduction (secondary prevention) of cardiovascular disease and stroke.* Retrieved February 9, 2005, from http://patients.uptodate.com/print.asp?print = true&file = screenpm/6460

4. Culleton, B. F., & Wilson, P. W. (2004). *Overview of the risk factors for cardiovascular disease.* Retrieved February 9, 2005, from http://patients.uptodate.com/topic.asp?file = cardiovasculardisease/36840&title= Cardiovascular+risk+factors

5. Health Canada. (2002). *Economic burden of illness in Canada, 1998*. Retrieved May, 26, 2005, from http://www.hc-sc.gc.ca/ahc-asc/media/nr-cp/2002/2002_ebic-femc_e.html

6. Canadian Association of Cardiac Rehabilitation. (2004). *Canadian guidelines for cardiac rehabilitation and cardiovascular disease prevention: enhancing the science, refining the art*. Winnipeg, MB: Author.

7. National Cholesterol Education Program. (2002). Third report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): Final report. *Circulation, 24*, 3142-3421.

8. Hayman, L. L., & Hughes, S. (2005). Preventing cardiovascular disease: Family matters. *Journal of Cardiovascular Nursing, 20*(1), 71-72.

9. Hennekens, C. H. (2004). *Primary prevention of cardiovascular disease and stroke.* Retrieved February 9, 2005, from http://patients.uptodate.com/ print.asp?print = true&file = screenpm/6460

10. Tanuseputro, P., Manue, D. G., Leung, M., Nguyen, K., & Johansen, H. (2003). Canadian cardiovascular outcomes research team: Reis factors for cardiovascular disease in Canada. *Canadian Journal of Cardiology, 19*(11), 1249-1259.

11. Ambrose, J. A., & Barua, R. S. (2004). The pathophysiology of cigarette smoking and cardiovascular disease: an update. *Journal of the American College of Cardiology, 43*(10), 1731-1737.

12. Wilson, K., Gibson, N., Willan, A., & Cook, D. (2000). Effect of smoking cessation after myocardial infarction: a meta-analysis of cohort studies. *Archives of Internal Medicine, 160*(7), 939-944.

13. Critchley, J., & Capewell, S. (2005). Smoking cessation for the secondary prevention of coronary heart disease (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

14. Jones, M. L. H., Granger, B. B., Short, L. M., & Taylor, M. C. (2004). A new response to heart disease in women: Promising disease management strategies offer state-of-the-science gender-based health promotion, screening, and intervention. *Nursing Management, 35*(7), 19-26.

15. Gluckman, T. J., Baranowski, B., Ashen, D., Henrikson, C. A., McAllister, M., Braunstein, J. B., et al. (2004). A practical and evidence-based approach to cardiovascular disease risk reduction. *Archives of Internal Medicine, 164*, 1490-1500.

16. Stead, L., & Lancaster, T. (2005). Group behaviour therapy programmes for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

17. Silagy, C., Lancaster, T., Stead, L., Mant, D., & Fowler, G. (2005). Nicotine replacement therapy for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

18. Abbot, N. C., Stead, L. F., White, A. R., & Barnes, J. (2005). Hypnotherapy for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

19. David, S., Lancaster, T., & Stead, L. F. (2005). Opioid antagonists for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

20. Hajek, P., & Stead, R. F. (2005). Aversive smoking for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

21. Park, E. W., Schultz, J. K., Tudiver, F., Campbell, T., & Becker, L. (2005). Enhancing partner support to improve smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

22. Ussher, M. H., West, R., Taylor, A. H., & McEwen, A. (2005). Exercise interventions for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

23. White, A. R., Rampes, H., & Ernst, E. (2005). Acupuncture for smoking cessation (Cochrane Review). *The Cochrane Library, 2*. Chichester, UK: John Wiley and Sons.

24. Bray, G. A. (2006). *Health hazards associated with obesity in adults.* Retrieved August 21, 2006, from http://patients.uptodate.com

25. Rowland, M. L. (1990). Self-reported weight and height. *American Journal of Clinical Nutrition, 52*(6), 1125-1133.

26. Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S., & Koplan, J. P. (2001). The continuing epidemics of obesity and diabetes in the United States. *Journal of the American Medical Association, 286*(10), 1195-1200.

27. Flegal, K. M., Carroll, M. D., Ogden, C. L., & Johnson, C. L. (2002). Prevalence and trends in obesity among US adults, 1999-2000. *Journal of the American Medical Association, 288*(14), 1723-1727.

28. Ardern, C. I., Katzmarzyk, P. T., Janssen, I., & Ross, R. (2003). Discrimination of health risk by combined body mass index and waist circumference. *Obesity Research, 11*(1), 135-142.

29. Booth, F. W., Gordon, S. E., Carlson, C. J., & Hamilton, M. T. (2000). Waging war on modern chronic diseases: Primary prevention through exercise biology. *Journal of Applied Physiology, 88*(2), 774-787.

30. Poirier, P., & Despres, J. P. (2003). Waist circumference, visceral obesity, and cardiovascular risk. *Journal of Cardiopulmonary Rehabilitation, 23*(3), 161-169.

31. Statistics Canada. (2002). *Canadian Community Health Survey: A first look.* (2002). Retrieved July 6, 2004, from http://www.statcan.ca/Daily/English/020508/d020508a.htm

32. US Centers for Disease Control and Prevention. (2002). *National diabetes fact sheet: General information and national estimates on diabetes in the Unites States, 2000.* Atlanta, GA: Author.

33. United Kingdom Prospective Diabetes Study Research Group. (1998). Intensive blood-glucose control with sulfonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes. *Lancet, 352*, 837-853.

34. Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., et al. (2003). The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Journal of the American Medical Association, 289*, 2560-2572.

35. Pescatello, L. S., Franklin, B. A., Fagard, R., Farquhar, W. B., Kelley, G. A., Ray, C. A., et al. (2004). *American College of Sports Medicine position stand: Exercise and hypertension, 36*(3), 533-553.

36. Touyz, R. M., Campbell, N., Logan, A., Gledhill, N., Petrella, R., & Padwal, R. (2004). The 2004 Canadian recommendations for the management of hypertension: Part III: Lifestyle modifications to prevent and control hypertension. *Canadian Journal of Cardiology, 20*(10), 55-59.

37. Lloyd-Jones, D., Wilson, P. W., Larson, M. G., Leip, E., Beiser, A., D'agatino, R. S., et al. (2003). Lifetime risk of coronary heart disease by cholesterol levels at selected ages. *Archives of Internal Medicine, 163*, 1966-1972.

38. Fodor, J. G., Frohlich, J. J., Genest, J. J. G., Jr., & McPherson, P. R. (2000). Recommendations for the management of treatment of dyslipidemia: Report of the Working Group on Hypercholesterolemia and Other Dyslipidemias. *Canadian Medical Association Journal, 162*(10), 1441-1447.

39. Cox, J. (1999). Optimizing disease management at a health care system level: The rationale and methods of the improving cardiovascular outcomes in Nova Scotia. *Canadian Journal of Cardiology, 15*, 787-796.

40. Harris, D. E., Gipson, G. W., & Pearson, T. A. (1998). Lipid lowering in a multidisciplinary clinic compared with primary physician management. *American Journal of Cardiology, 81*, 929-933.

41. Mosca, L., Merz, N. B., Blumenthal, R. S., Cziraky, M. J., Fabunmi, R. P., Sarawate, C., et al. (2005). Opportunity for intervention to achieve American Heart Association guidelines for optimal lipid levels in high-risk women in a managed care setting. Circulation, *111*(4), 488-493.

42. Yates, S., Annis, L., Pippins, J., & Walden, S. (2001). Does a lipid clinic increase compliance with National Cholesterol Education Program treatment guidelines? Report of a case-matched control study. *Southern Medicine, 94*, 907-909.

43. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. (2001). *Executive summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol.* Retrieved February 25, 2005, from http://www.nhlbi.nih.gov/guidelines/cholesterol/atp_iii.htm

44. Joffres, M. R., Chadirian, P., Fodor, J. G., Petrasovitis, A., Chockalingam, A., & Hamet, P. (1997). Awareness, treatment, and control of hypertension in Canada. *American Journal of Hypertension, 10*(10), 1097-1102.

45. Goldberg, R. J., Currie, K., White, K., Brieger, D., Steg, P. G., Goodman, S. G., et al. (2004). Six-month outcomes in a multinational registry of patients hospitalized with an acute coronary syndrome (The Global Registry of Acute Coronary Events [GRACE]). *American Journal of Cardiology, 93*(3), 288-293.

46. Hemmelgarn, B. R., Zarnke, K. B., Campbell, N. R., Feldman, R. D., McKay, D. W., McAlister, F. A., et al. (2004). The 2204 Canadian Hypertension Education Program recommendations for the management of hypertension: Part I: Blood pressure measurement, diagnosis, and assessment of risk. *Canadian Journal of Cardiology, 20*(1), 31-40.

47. DeBusk, R. F., Miller, N. H., Superko, H. R., Dennis, C. A., Thomas, R. J. , Lew, H. T., et al. (1994). A case-management system for coronary risk factor modification after acute myocardial infarction. *Annals of Internal Medicine, 120*(9), 721-729.

48. MacLean, D. R., Petrasovitis, A., Connelly, P. W., Little, J. A., & O'Connor, B. (1999). Impact of different blood lipid evaluation and treatment guidelines on the proportion of Canadians identified and treated for elevated blood cholesterol. *Canadian Journal of Cardiology, 15*, 445-451.

49. Murchie, P., Campbell, N. C., Ritchie, L. D., Simpson, J. A., & Thain, J. (2003). Secondary prevention clinics for coronary heart disease: Four-year follow up of a randomized controlled trial in primary care. *British Medical Journal, 326*(7380), 84-87.

50. Lai, S. C., & Cohen, M. N. (1999). Promoting lifestyle changes. *American Journal of Nursing*, 99(4), 63-67.

51. Basler, H. D. (1995). Patient education with reference to the process of behavioural change. *Patient Education Counsellor, 26*, 93-98.

52. Vermeire, E., Hearnshaw, H., Van Royen, P., & Denekens, J. (2001). Patient adherence to treatment: Three decades of research. A comprehensive review. *Journal of Clinical Pharmacy and Therapeutics, 26*(5), 331-342.

53. Lorig, K. R., & Holman, H. (2003). Self- management education: History, definition, outcomes, and mechanisms. *Annals of Behavioral Medicine, 26*(1), 1-7.

54. Wheeler, J. R., Janz, N. K., & Dodge, J. A. (2003). Can a disease self-management program reduce health-care costs? The case of older women with heart disease. *Medical Care, 41*(6), 706-715.

55. Jackson, L., Leclerc, J., Erskine, Y., & Linden, W. (2005). Getting the most out of cardiac rehabilitation: A review of referral and adherence predictors. *Heart, 91*(1), 10-14.

56. Ades, P. A., Waldman, M. L., McCann, W. J., & Weaver, S. O. (1992). Predictors of cardiac rehabilitation in older coronary patients. *Archives of Internal Medicine, 152*(5), 1033-1035.

57. Turner, S. C., Bethell, J. J., Evans, J. A., Goddar, J. R., & Mullee, M. A. (2002). Patient characteristics and outcomes of cardiac rehabilitation. *Journal of Cardiopulmonary Rehabilitation, 22*(4), 253-260.

58. Leibowitz, M., Regess, O., Manor, D., Bental, T., & David, D. (2005). Nonparticipation in cardiac rehabilitation and analysis utilizing classification and regression trees. *Journal of Cardiopulmonary Rehabilitation, 25*, 88-92.

59. Haynes, R. B., McDonald, H., Garg, A. X., & Montague, P. (2005). Interventions for helping patients to follow prescriptions for medications (Cochrane Review). *The Cochrane Library, 25*, 1-52.

60. Petrie, K. J., Weinman, J., Sharpe, N., & Buckley, J. (1996). Role of patients' view of their illness in predicting return to work and functioning after myocardial infarction: Longitudinal study. *British Medical Journal, 312*(7040), 1191-1194.

61. Gavic, A. M. (2005). Addressing the problem of cardiac rehabilitation program distribution. *Journal of Cardiopulmonary Rehabilitation, 25,* 86-87.

62. Dunbar-Jacob, J., Bohachick, P., Mortimer, M. K., Sereika, S. M., & Foley, S. M. (2003). Medication adherence in persons with cardiovascular disease. *Journal of Cardiovascular Nursing, 18*(3), 209-218.

63. Grace, S. L., Abbey, S. E., Shnek, Z. M. (2002). Cardiac rehabilitation II: Referral and participation. *General Hospital Psychiatry, 24*, 127-134.

64. Brink, P. J., & Wood, M. J. (1998). Reliability and validity of measurement. In P. J. Brink & M. J. Wood (Eds.), *Advanced design in nursing research* (2nd ed., pp. 173-198). Thousand Oaks CA: Sage.

65. Gray, J. (2003). Dyslipidemias. In J. Gray (Ed.), *Therapeutic choices* (4th ed., pp. 241-249). Ottawa, ON: Canadian Pharmacists Association.

APPENDIX A: TRACKING CARDIOVASCULAR RISK FACTORS

Cardiovascular Risk Reduction Clinic, University of Alberta Hospital
Cardiovascular Risk Factors

Age: _____ Hypertension: _____ Diabetes: _____

Smoking:
- ☐ Never
- ☐ Prior quit x _____yrs
- ☐ Current _____ppd x _____yrs

Family Hx of Vascular Disease: _____
Elevated Lp(a): (nl <0.30 g/L) _____
Elevated Homocysteine: (nl 2.1µmol/L) _____
Known Vascular Disease:
- ☐ Nil _____
- ☐ MI _____
- ☐ Angina _____
- ☐ PTCA _____
- ☐ CABG _____
- ☐ TIA _____
- ☐ Stroke _____
- ☐ PVD _____

Total # of Risk Factors: _____
10-year risk of CHD event (%): _____
ASA (Yes/No): _____

Other Medications

Name	Dose (mg/d)	Name	Dose (mg/d)

(G. Pearson, personal communication, March 15, 2005)

APPENDIX B: CARDIOVASUCLAR RISK REDUCTION CLINIC DATA SHEET

CLINIC DATE		
Height		
Weight		
BMI		
BP/HR	/	/
Waist Circumference		
Hypertension		
Smoker (current/prev/never)		
Diabetic		
Family History		
Transplant (type)		
Known Vascular Dz		
MEDICATIONS (mg/d)		
Lipid Medications		
Drug Allergy/ Intolerance		
LIPID PROFILE DATE		
Total Cholesterol Target:		
LDL Target:		
HDL Target:		
Triglycerides Target:		
TC/HDL Ratio		
Lp(a)		
Hcy (<12.1 µmol/L)		
TSH (0.20-6.10 mU/L)		
Fasting Glucose		
HbA1c		
K^+		
Creatinine		
Albumin/Creatinine Ratio		
ALT (<50 U/L)		
AST (<40 U/L)		
CK (<180 U/L)		
hs-CRP		
Uric Acid		
Apo B		

(Amended form; G. Pearson, personal communication, March 15, 2005)

APPENDIX C: REQUEST FOR ETHICS COMMITTEE APPROVAL

Health Research Ethics Board
Request for Ethics Review Form

Section A: General Information

A1. Project Title			
Title of Project: Cardiovascular Risk Factors			
A2. Applicant Information			
Name: Colleen Norris Title: Associate Professor Department: Faculty of Nursing, University of Alberta			
Mailing Address: 36 McGill St			
City & Province: Red Deer, Alberta	Postal Code: T4R 1S3	Phone: 403-343-2759	Fax: 403-343-3206
E-mail Address: colleen.Norris@ualberta.ca			
Signature:			Date: 05/03/15
A4. Authorizing Signature			
Indication of Department Support for the Implementation of the Project.			
Name of Dept. Chair, Assoc. Dean of Research, or Supervisor:			
Title:			
Signature:			Date:
A5. Co-Investigators / Thesis Committee			
Is this project for a graduate thesis? (*) Yes ( ) No If yes, please provide the names, departments, and phone numbers of your thesis committee.			
Name: Sandra Engi	Department/Program: Nursing	Phone: 403-343-2759	
A6. Expedited Review			
If the study procedures are **LIMITED** to any of the following, please check (√):			
	Analysis of blood, urine, or any other biological specimen already collected.		
	Examination of patient, medical, or institutional records.		
	Modification of a previously approved protocol (specify title and approval date):		
*	Secondary analysis of data.		
	Use of biological specimens normally discarded.		

A7. Type of Investigation			
Which one of the following best describes the type of investigation proposed? Check (√) more than one if appropriate.			
	Clinical Trial		Multi-centre Trial
	Drug Study		Pilot Study
	Epidemiological Study		Qualitative Study
	First Application in Humans		Technology Assessment / Development
	Sequel to Previously Approved Project (specify title and approval date):		
*	Other (specify): Non experimental retrospective correlational design		
A8. Site of Research			
Where will the research be conducted? Check (√) more than one if appropriate. Specify the area/department/program.			
*	University of Alberta Hospital:		
University of Alberta Sites:			
*	Specify (e.g. Corbett Clinic): Lipid reduction clinic		
Letters of Support:			
(*) Pending () Attached () Not Applicable			
A9. Funding / Budget			
How is the project funded? Please check (√) the appropriate box.			
	Funding approved; specify source(s):		
	Funding pending; specify source(s):		
*	No external funding required.		
Budget			
*	Please check here (√) that you have attached a budget summary. The summary must include details of investigator payments and recruitment incentives (if present). Please attach the budget as an appendix to the form.		
A10. Remuneration			
Are any of the investigators involved receiving any directs personal remuneration or other personal or family financial benefits (either direct or indirect) for taking part in this investigation?			
	Yes. If so, append a letter detailing these activities. Please attach this letter to your budget summary.		
*	No.		

A11. Safety Approvals					
Please check (√) whether or not this study requires any of the following safety approvals. If a safety approval is needed, please indicate whether the approval documentation is pending or attached as an appendix to this form.					
Biohazardous Materials:					
*	Not Applicable		Pending		Attached
Electromechanical:					
*	Not Applicable		Pending		Attached
Health Protection Branch or Other Canadian Federal Agency:					
*	Not Applicable		Pending		Attached
Radiation:					
*	Not Applicable		Pending		Attached

Section B: Details of Project

Description of the Project
B1. Provide a clear statement of the purpose and objectives of the project. The purpose of this study is to explore the relationship between patient characteristics and successful risk reduction among patients attending a lipid reduction clinic at the University of Alberta Hospital between the years 2000-2004.
B2. State the hypotheses and/or research questions. What are the patient characteristics of successful cardiovascular risk reduction?
B3. Briefly summarize past human and/or animal research that has lead to this project. Modifiable risk factors for cardiovascular disease (CVD) are largely preventable and are mostly a consequence of lifestyle choices; 55% of reduction in cardiovascular disease mortality is due to risk-factor reduction. Literature demonstrates that characteristics of patients who are successful at risk reduction require further definition in order to define effective programming.
Description of Sample/Population
B4. Describe the numbers and type(s) of subjects to be included. If appropriate, specify the number of subjects in each study group. Provide a rationale for the sample size and include sample size calculations where appropriate. Once participants are screened for inclusion and exclusion criteria the eligible participants will then be randomized for inclusion utilizing SPSS software. This randomization will be done to help assure a representative sample so that the results can then be further generalized to the population (Brink & Wood[64]). Sample size will be 520 patients. This sample size was determined utilizing guidelines provided in regards to sample size calculation for logistical regression. The sample must include 10 participants for each dichotomous variable and 20 participants for each level of continuous, ordinal or interval variable (William Midozi - Data Analyst., personal communication, March, 10, 2005). As previously described this study is examining a total of 7 independent variables and 1 dependent variable. There are a total of 3 dichotomous variables, including the dependent variable

B5. List any subject inclusion/exclusion criteria. For the purposes of this study all individuals between the ages of 20-99 will be included. Patients with heart failure, end stage renal disease, and Type 1 diabetes will be excluded as these conditions add a level of complexity that is not appropriate for the purpose of this study.
Description of Research Procedures
B7. Provide a summary of the design and procedures of the research. Provide details on the methods of data collection and data analysis, time commitment for the subjects etc. Please note that any and all study measures need to be appended to the copies of the research / grant proposal (e.g. questionnaire, interview guides, rating scales etc.). The data will be analyzed using logistical regression to determine if there is a relationship between certain variables and successful risk reduction. The Independent variables will include: age, gender, income level, BMI on admission, smoking, and Framingham Risk score. The dependent variable successful risk reduction is the attainment or non attainment of target LDL levels as defined by the NCEP guidelines.
B8. Which treatments or procedures are additional to those required for standard patient care? NA
B9. If the procedures include a blind, under what conditions will the code be broken and what provisions have been made for this? Who will have the code? NA
Obtaining Consent
B10. Clearly detail who will be recruiting subjects and obtaining consent, and the procedures for doing this. If appropriate specify whether subjects will be randomly assigned to groups before or after consent has been attained. Informed consent was obtained from patients for participation in the lipid reduction clinic. Part of the consent involved the use of data for research. There is no separate consent form for the clinical database, due to its nature of being a consolidation of information that was gathered in the course of the clinical management of the patient with the primary intention of being used to provide ongoing clinical care. Any study, using data from this database, is required to be submitted to the University of Alberta Research Ethics Committee for approval. (G. Pearson, personal communication, March, 16, 2005)
B11. Specify methods for dealing with groups identified in #B6. If the subjects are not able/competent to give fully informed consent, who will consent on their behalf? NA
B12. If the subjects will be offered compensation for participating in the research, provide details. Specify the amount, what the compensation is for, and how payment will be determined for subjects who do not complete the study. NA
B13. Do any of the procedures include the use of deception or partial disclosure of information to subjects? If yes, provide rationale for the deception or partial disclosure. Describe the procedures for (a) debriefing the subjects and (b) giving them a second opportunity to consent to participate after debriefing. NA

Recruitment Aids/Information Letters/Consent Forms					
B14. Are you planning to use any recruitment aids such as posters, newspaper advertisements, radio announcements, or letters of invitation? If so, please indicate the reading level of each aid and check (√) if it has been attached to the form as an appendix.					
Recruitment Aid #1 – Specify (e.g. poster, letter etc.):					
*	Not Applicable		Reading Level		Attached
Recruitment Aid #2 – Specify:					
*	Not Applicable		Reading Level		Attached
Information Letter #1 – Specify (e.g. Letter for interviews, focus groups etc.):					
*	Not Applicable		Reading Level		Attached
Information Letter #2 – Specify:					
*	Not Applicable		Reading Level		Attached
Consent Form #1 – Specify (e.g. Consent for interview, focus group etc.):					
*	Not Applicable		Reading Level		Attached
Consent Form #2 – Specify:					
*	Not Applicable		Reading Level		Attached
B15. What steps have been taken to make the recruitment aids, information letters, and consent forms comprehensible to the person(s) giving consent?					
Risks and Benefi					
B16. What are the benefits of the proposed research for the subject and/or for scientific knowledge in general? This information can be used to evaluate current programming and assist in the development of new strategies to target members of the population not currently attaining successful risk reduction.					
B17. What adverse effects may result from the research? How will adverse effects be dealt with? Please note that adverse effects are not limited to physical risks, but include psychological, emotional, and spiritual risks as well. NA, utilizing secondary dataset					
Privacy and Confidentiality					
B18. What steps will be taken to respect the privacy of the subjects and protect confidential data? No use of personal identifying information in the secondary data collection. Results will be published with numbers only					
B19. Identify any agencies or individuals who will have access to confidential data now or in the future. University of Alberta Hospital cardiac research division/University of Alberta, Faculty of Nursing.					
B20. Do you anticipate any secondary analysis of the data? Please note that any secondary analysis requires further research ethics approval. This is a secondary analysis of data					

APPENDIX D: CARDIOVASCULAR RISK REDUCTION

CLINIC FOLLOW-UP FORM

Cardiovascular Risk Reduction Clinic
Follow-Up Visit

Dear Dr. _____ Date: _____
RE: _____ Last Seen: _____

Current Treatment for Dyslipidemia

Diet: _____
Exercise: _____
Lipid Medications: _____
Side Effects: _____
Prior Intolerances: _____
Other Symptoms: _____

Coronary Risk Factors/Other Medications: Please See Over

Lab Summary (Most Recent Results)

Date	TChol	LDL	HDL	TG	TC/HDL	ALT	CK	Lipid medication(s)

Other Lab Results: _____

Physical Exam: BP: _____ Weight: _____
Other: _____
_____

Impression/Plan:

Recommended Lipid Targets: LDL <____ TG <____ TC/HDL <____

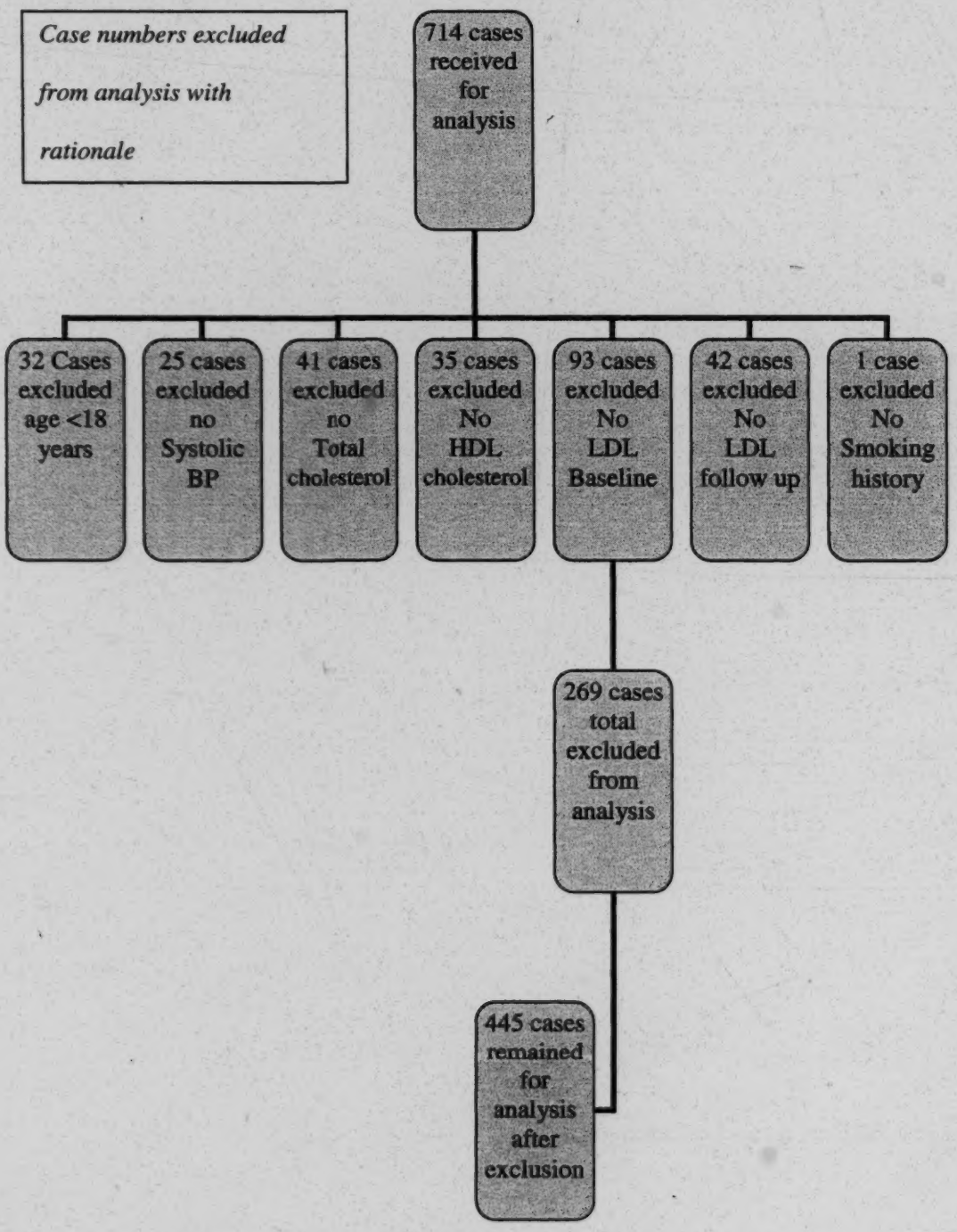
APPENDIX E: DETERMINATION OF FRAMINGHAM RISK SCORE

Management of Dyslipidemia

Assess risk using Framingham data				
Step 1	Low risk 10 yr risk <10%	Moderate risk 10 yr risk 10-20%	High risk 10 yr risk 20-30%	Very high risk 10 yr risk ≥ 30% or diabetes or clinically evident CHD
Step 2	Target lipid levels LDL-C <5mmol/L TC/HDL-C <7 TG< 3 mmol/L	Target lipid levels LDL-C <4mmol/L TC/HDL-C <6 TG< 2	Target lipid levels LDL-C <3mmol/L TC/HDL-C <5 TG< 2 mmol/L	Target lipid levels LDL-C <2.5mmol/L TC/HDL-C <4 TG< 2 mmol/L
Step 3	Non Pharmacologic choices	Non Pharmacologic choices	Both pharmacologic and nonpharmacologic choices	Both pharmacologic and nonpharmacologic choices
Step 4	Add pharmacologic choices if target not met after 6 months	Add pharmacologic choices if target not met after 6 months		

(Gray,[65] pp. 244-245)

APPENDIX F: FLOW CHART OF CASE EXCLUSION WITH NUMBERS AND RATIONALE


Case numbers excluded from analysis with rationale
714 cases received for analysis
32 Cases excluded age <18 years
25 cases excluded no Systolic BP
41 cases excluded no Total cholesterol
35 cases excluded No HDL cholesterol
93 cases excluded No LDL Baseline
42 cases excluded No LDL follow up
1 case excluded No Smoking history
269 cases total excluded from analysis
445 cases remained for analysis after exclusion